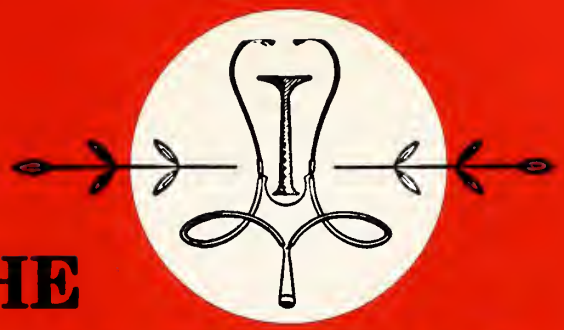
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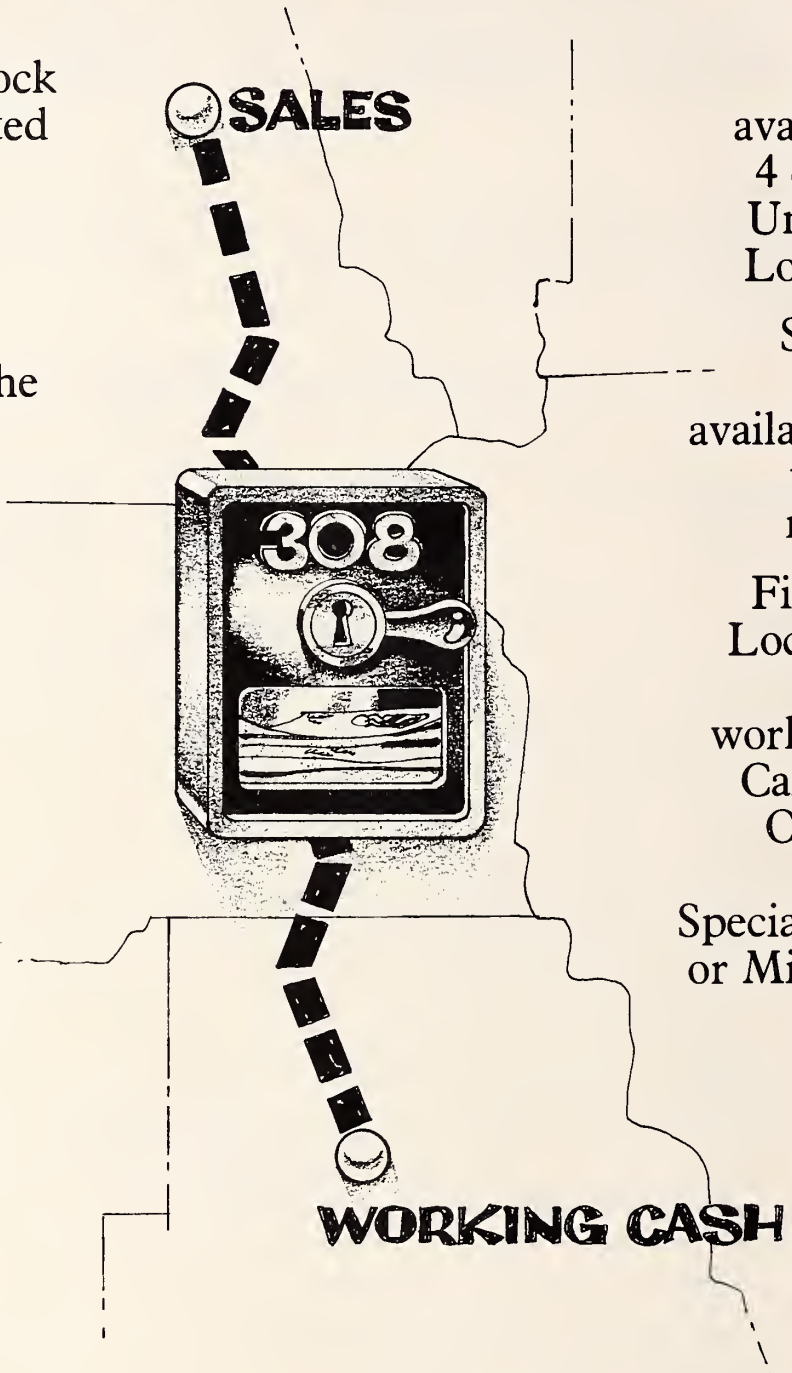
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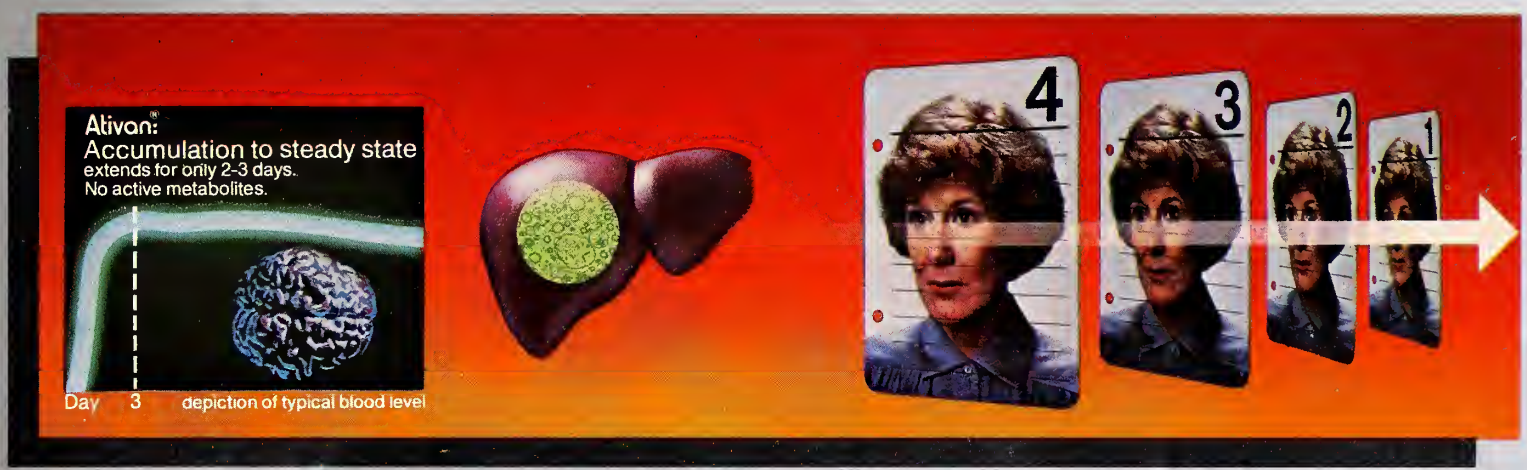
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Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

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NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

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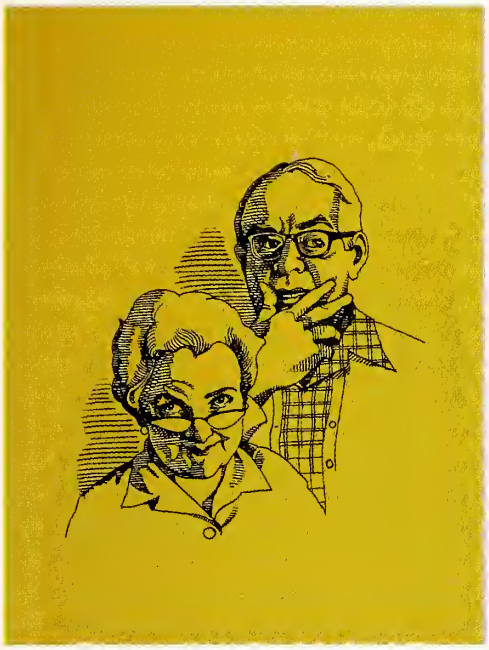
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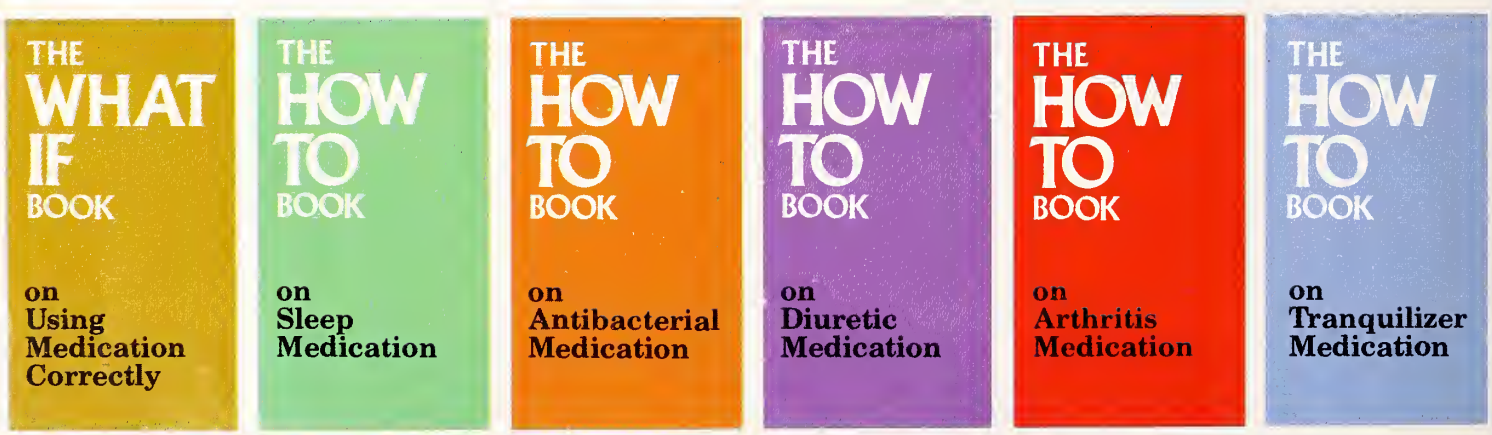
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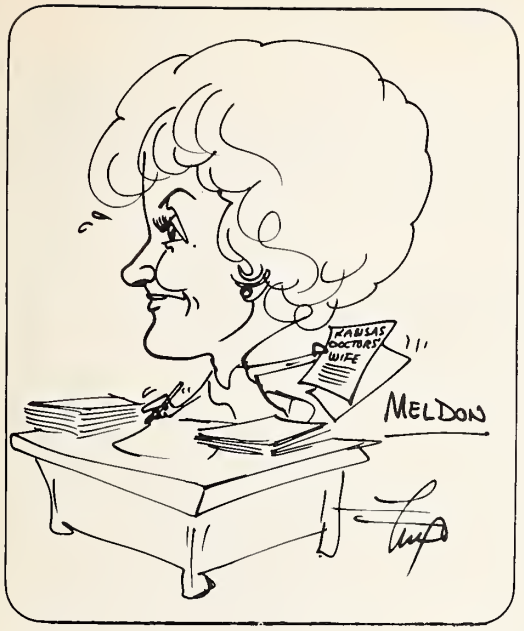
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An Open Letter to Kansas Physicians

Ed. Note: Meldon Laury, Auxiliary President, has relinquished her space in this month's *Journal* to the American Association of Medical Assistants-Kansas Society.

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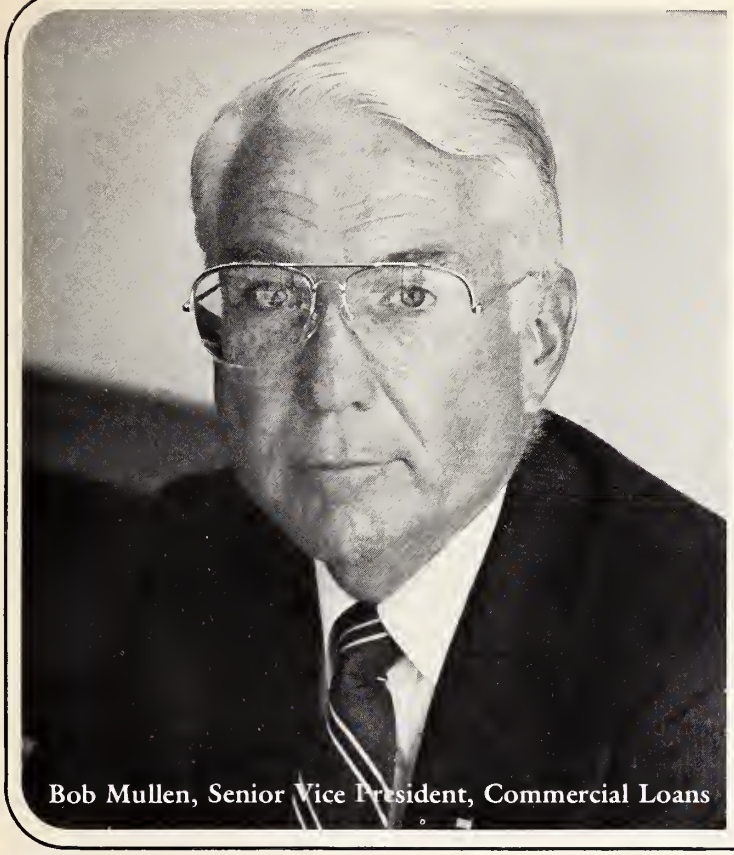
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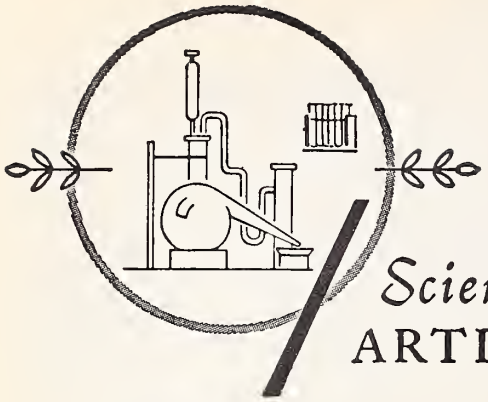
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Hyperbilirubinemia and Mono

Marked Hyperbilirubinemia in Infectious Mononucleosis

WILLIAM F. CATHCART-RAKE, M.D.,* *Salina*

THE INCIDENCE of jaundice in infectious mononucleosis has varied from 6-14 per cent in previously reported series.[1-5] Hyperbilirubinemia is usually mild and of short duration. Reported here is a case of marked hyperbilirubinemia in a young male with infectious mononucleosis.

Case Report

A 19-year-old male was admitted to Asbury Hospital in Salina on February 9, 1982, with a four day history of intermittent fever and chills, a two day history of profound fatigue, and a one day history of nausea and vomiting. He had noticed dark urine for four days prior to admission, and on the day prior to admission he was told that his sclera were yellow in color. He had had no previous serious illnesses, had been taking no medication, and denied excessive alcohol use.

On admission the patient was deeply jaundiced. Cervical lymphadenopathy and evidence of pharyngitis were initially absent, but did develop two days after admission. Examination of the abdomen revealed slight tenderness in the right upper quadrant; the liver edge was palpated at the right costal margin and the total liver span was 8 cm. The spleen tip was palpated 4 cm below the left costal margin. Results of the remainder of the physical examination were unremarkable.

* Clinical Assistant Professor of Medicine, The University of Kansas School of Medicine-Wichita.

Address reprint requests to Dr. Cathcart-Rake, Mowery Clinic, 737 E. Crawford, Salina KS 67401.

Admission hematocrit was slightly low at 36.2%; leukocyte count was 14,500 with 69% lymphocytes, 18% polymorphonuclear leukocytes, 7% band forms, and 6% monocytes. All lymphocytes had atypical morphology. Serum alkaline phosphatase was 204 U/L; SGOT, 264 U/L; LDH, 615 U/L; and total bilirubin, 12.9 mg/dl. Presumptive heterophil antibody was positive at a titer of 1:1797, and was unchanged after exposure to Forssman antigen; absorption with beef red cells decreased the titer to 1:224. Hepatitis B surface antigen and hepatitis A antibody were negative. On the day following admission his total bilirubin was 11.7 mg/dl with a direct fraction of 7.4 mg/dl.

Subclinical hepatitis is common in infectious mononucleosis. Jaundice occurs in approximately 10 per cent of cases. The association of infectious mononucleosis and hyperbilirubinemia (total serum bilirubin level > 8.0 mg/dl) is rare, but mono must be considered in the intensely jaundiced young patient.

The patient received intravenous fluids until nausea and vomiting subsided. His symptoms gradually resolved, and results of liver function studies gradually returned to normal following admission. By February 13, he was able to tolerate an oral diet and was discharged from the hospital. On February

19, he was essentially asymptomatic. He had large anterior cervical lymphadenopathy and a barely palpable spleen tip. On this date, SGOT, alkaline phosphatase, and LDH were normal; bilirubin was 1.9 mg/dl; and hematocrit was 38.9%.

Discussion

Jaundice in infectious mononucleosis can occur either from hemolysis mediated by a cold agglutinin or, more frequently, from hepatitis. Hepatitis is believed to be common in infectious mononucleosis because liver function tests are often abnormal; however, clinical manifestations of hepatitis are infrequent. Liver biopsies in patients with infectious mononucleosis have revealed infiltration of portal regions with mononuclear cells, areas of scattered mononuclear necrosis, sinusoidal invasion by monocytes, and proliferation of Kupffer cells.[6] The hyperbilirubinemia seen in this case was probably secondary to a combination of hepatitis and mild hemolysis.

Marked hyperbilirubinemia in infectious mononucleosis is thought to be extremely rare. In Bernstein's classic review in 1940 the highest reported bilirubin was 8.0 mg/dl.[7] The actual incidence of marked hyperbilirubinemia (total bilirubin level of 8.0 mg/dl or greater on at least one occasion) is difficult to determine because of omission of data on serum bilirubin levels in those reports of large numbers of patients. In a series of 69 patients Gelb *et al.*[3] found no patients with a total bilirubin level of 8.0 mg/dl or more. Futterweit[2] and Shuster and Ognibene[8] documented marked hyperbilirubinemia in one of 51 patients and one of 46 patients, respectively. *Table I* is a compilation of previously reported patients with total serum bilirubin levels of 8 mg/dl or greater at some time during the course of their illness.

It is speculated that marked hyperbilirubinemia in infectious mononucleosis occurs more frequently than originally thought, and probably more frequently than is reported. However, marked elevation of bilirubin levels, whether as a result of hepatitis, hemolysis, or a combination of both, undoubtedly remains extremely rare compared to the incidence of the disease. Fortunately, infectious mononucleosis hepatitis has not been reported to proceed to chronic hepatitis and only rarely has acute hepatitis from infectious mononucleosis resulted in death.[18-20] Nevertheless, infectious mononucleosis must be considered in the intensely jaundiced young patient with hepatitis.

Acknowledgement

Norton J. Greenberger, M.D., assisted in the preparation of this manuscript.

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(Continued on page 38)

TABLE I
PREVIOUSLY REPORTED PATIENTS WITH SERUM BILIRUBIN ≧ 8 mg/dl

Reference	*# of Patients Reported*	*Highest Total Serum Bilirubin(s) (mg/dl)*	*Evidence of Hemolysis*
Abrams[9]	1	27.5	ND*
Carter[10]	1	22.5	ND
Corr & Scian[11]	1	19.4	Yes
Davies *et al.*[12]	1	31.0	ND
Futterweit[2]	1	8.7	ND
Gold[13]	1	23.3	ND
Hoaglund & McCluskey[14]	1	8.0	ND
Houk & McFarland[15]	1	17.2	Yes
Madigan *et al.*[16]	1	38.5	Yes
Samuels[17]	3	37.0, 17.9, 10.7	Yes (all cases)
Shuster & Ognibene[8]	1	10.5	ND

* ND = not determined or insufficient data to determine if hemolysis occurred.

Hemoccult Screening

Diagnosis of Colorectal Neoplasms

DONALD D. MOELLER, M.D., *Kansas City, Kansas*

COLORECTAL CANCER has caused more deaths during the last 40 years than any other cancer in the United States.[1] There is now evidence that cancer of the colon and rectum can be diagnosed at an earlier stage by doing annual Hemoccult screening tests supplemented by sigmoidoscopy every three years.[2] Presented here are results of annual Hemoccult screening in a small group of patients who have been followed in the author's private practice.

Methods

From 1977 to 1981, a number of people over age 40 years who were asymptomatic or had stable gastrointestinal symptoms were enlisted to participate in the Hemoccult screening program. Patients were advised to adhere to a diet high in fiber (8-10 items of high fiber food daily) and free from red meat (fish and fowl were allowed) for six days. During this time period neither aspirin nor vitamin C was allowed. During the last three days of the diet patients were instructed to obtain a small sample from each end of a stool for a total of six specimens. The Hemoccult cards were either brought to this office or mailed. The cards were developed by standard procedures.

Results

A total of 57 patients were entered into this study. Thirty-nine were females with an average age of 65 years (varying from 45 to 90 years). Eighteen males had an average age of 58 years (varying from 44 to 76 years). Diagnoses were determined from a chart review and are listed in *Table I*. Only two patients had four annual Hemoccult screening tests; seven had three annual tests; 21 had two, and 27 had one.

Many of the patients had tests of their gastrointestinal tract before entering the study. All tests were done during the 1977-1981 period. Twenty-three barium enemas, 24 sigmoidoscopies, and seven colonoscopies were performed. Eleven patients had no gastrointestinal tests except for Hemoccult screening (although they were advised to have a sigmoidoscopy every three years). All patients with potentially bleeding lesions (such as peptic ulcers) were treated maximally before they underwent Hemoccult screening.

There were a total of 97 Hemoccult screening tests performed on these 57 patients. Only one patient had positive results from the Hemoccult test; in the fourth year of Hemoccult screening one of his six stool specimens was positive. Barium enema showed an "apple core" lesion of the proximal transverse colon. He underwent right colectomy and is alive and free of disease two years later. This single positive test in 57 patients represented 1.8 per cent of the study group. The single positive in 97 Hemoccult screenings equals 1 per cent of the total tests.

Fifty-seven asymptomatic or stable symptomatic patients over age 40 years were followed with annual Hemoccult screening for one to four years. Only one patient had a positive Hemoccult test result; he was found to have carcinoma of the colon. One per cent of Hemoccult tests yielded positive results and 1.8 per cent of the patients were positive. No false positive results were discovered. The cost of diagnosis of this single case of cancer of the colon was calculated to be $1,254.

The cost for diagnosing this cancer of the colon was calculated by multiplying 97 — the number of Hemoccult screenings done — times $12 — the cost of each Hemoccult screening. (The initial kit or envelope of material costs $3 to the patient and each double-boxed card costs $3 for development and interpretation. Most insurance companies will pay for the cost of interpretationof these tests.) Thus, 97 times $12 equals $1,164. If one adds the cost of a barium enema (estimated at $90), the total cost for diagnosis of this single carcinoma of the colon was $1,254. The reason for including only one barium enema in the cost was that this was the only test that evolved directly from the Hemoccult screening program.

Discussion

A simple and accurate screening test for detecting occult blood in stools has long been sought. Irons

TABLE I
DIAGNOSES

Well patients	9
Schatzki's ring	1
Esophageal stricture	2
Esophagitis	1
Esophageal webb	1
Gastritis	2
Gastric bezoar	1
Gastric ulcer	1
Peptic ulcer disease	4
Post-gastrectomy diarrhea	1
Gallstones	1
Cirrhosis	1
Hyperplastic colon polyps	1
Irritable bowel syndrome	7
Diverticulosis of the colon	12
History of colon polyps	1
Angioid dysplasia of the colon	1
Hypertension	12
Aortic stenosis	1
Coronary artery disease	14
Seizure disorder	1
Diabetes mellitus	1
Renal calculi	1
Hypothyroidism	1
Iron deficiency anemia	1
Obesity	1
Chronic bronchitis	1
Migraine headaches	1

and Kirsner summarized the state of the art for detection of stool occult blood in 1965.[3] They concluded that benzidine and orthatolidine were very sensitive (detecting 0.5-9.0 cc of blood in the gastrointestinal tract) but had an incidence of false positive reactions of 75-100 per cent. The guaiac test could detect 2-50 cc of blood in the alimentary tract and had a false positive rate of 25 per cent with normal diet.

A paper by Greegor in 1967 indicated that in a review of his cases of silent (asymptomatic) colorectal carcinomas, all seven had positive guaiac tests on at least one of three consecutive daily stools (with a minimum of two separate specimens obtained from each stool).[4] However, the overall number of asymptomatic cases was 23 per cent positive for Hemoccult testing. Greegor conceded that this was an inordinate number of people who required referral to the radiologist for barium enemas.

In 1971, Greegor reported that by utilizing a meat-free high fiber diet, he could reduce the number of false positive Hemoccult tests to 5 per cent.[5]

In 1976, Winawer *et al.* presented an excellent summarization of the background and progress in screening stools of people over the age of 40 years for occult blood.[6]

By 1980, there had been numerous clinical reports on this type of asymptomatic detection of colorectal cancers. The largest study was reported out of Germany.[7] Approximately 3.5 million West Germans participated in a Hemoccult screening program. Of those positive, 4.7 per cent and 10.7 per cent of females and males respectively were found to have carcinoma of the colon or rectum.

In 1980, the American Cancer Society recommended that all people age 50 years or older should undergo annual Hemoccult screening, supplemented by sigmoidoscopy every three years.[2] One of the main justifications for this recommendation came from the ongoing Hemoccult screening program at the University of Minnesota. Of those patients screened annually by Hemoccult, Duke's A lesions were found in 65 per cent, whereas in controls who elected not to have Hemoccult screening, Duke's A lesions were found in only 15 per cent.

The principles of Hemoccult screening are as follows:

1. Asymptomatic patients over the age of 40 or 50 years (age 40 yrs preferable);
2. A high fiber diet free from red meat (no beef, pork, venison or elk, but fish and fowl allowed) for six days with no aspirin or vitamin C medication allowed;
3. Testing of two samples from stools on days 4, 5, and 6; and
4. Specimens tested within a 3-4 day period from collection.

The literature and the author's experience indicate that when these principles are followed:

- There will be a 1.5-3.5 per cent positive rate; *i.e.*, results for one to three of each 100 patients tested will be positive.
- Of each patient with positive results of Hemoccult screening, approximately 50 per cent or one-half will have a lesion in the colon that could have been the source of occult blood (cancer, polyp, colitis, angioid dysplasia).
- Earlier detection of colorectal neoplasms is possible.
- Improved cure rate for colorectal cancer has not been shown.
- Relatively good cost/benefit ratio has been shown in the study presented.

References

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(Continued on page 29)

Nausea and Vomiting

Its Management in Cancer Chemotherapy

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MANAGING nausea and vomiting (N&V) that often accompanies cancer chemotherapy is a major issue in medicine today. As the number of chemotherapy patients increases and potent new anti-cancer agents increase the vomiting potential, N&V control becomes more difficult.

While oncology specialists scan the research horizon for a major drug break-through to solve the growing problem, one avenue to the improvement in anti-N&V treatment may have been detoured — falling back on the art of medicine. The artful physician, with a selection of one or two drugs from a limited existing group, might significantly increase the current 40-50 per cent success rate.

The principle of interdisciplinary cooperation can assist in the improvement of control of nausea and vomiting. The oncologist is the acknowledged master in deployment of antineoplastic drugs. N&V management may be more effectively directed by a neuropsychiatrist.

The neuropsychiatrist brings to the issue two therapeutic approaches — psychotherapy and psychopharmacology — that can be of assistance to the oncologist. The neuropsychiatrist's forte traditionally has been psychodynamic psychotherapy, along with skills in psychonosology, *i.e.* the diagnosis of mental and emotional states. Going further, the psychic state of the cancer chemotherapy patient is of critical importance in the outcome of his/her treatment. In fact, as implied by Chang,[1] it may be the pivotal issue.

All patients facing a cancer chemotherapy regimen should have the option of a psychiatric consultation. For many, brief supportive psychotherapy may obviate the need for antiemetic medication or at least significantly reduce anti-N&V medication dosages.

Fear, anxiety, depression, anger, frustration, and denial are common emotional responses for an individual who has been informed that s/he has cancer. Disregard for the emotional makeup and mental status of the patient may increase the risk of negative results and effects, *e.g.*, postponement of chemotherapy by the patient and "rejection" of medication via emesis. Psychodynamically speaking, regurgitation of the means of effecting a cure — the medication — may be a way of denying the existence of the dreaded disease. When the particular affect — anxiety, anger — is strong enough, limbic and diencephalic input may be sufficient to stimulate the vomiting center (VC) and thus produce emesis.

Nausea and vomiting associated with cancer chemotherapy is of significant concern to oncologists and their patients. The psychological component of the patient's illness is as important as the cancerous lesion or condition. A neuropsychiatrist, in cooperation with the oncologist, can often assist in the effective management of nausea and vomiting caused by cancer chemotherapy.

Once a psychodynamic formulation has been made, the neuropsychiatrist can help the patient to work through the emotional side of the illness. The classic, long-term "talk therapy" of the psychoanalyst can be effectively condensed into several sessions to meet the needs of a cancer patient. Adjunctive therapies, such as hypnosis and relaxation, may be useful for some patients. When depression is a significant factor, antidepressant medication may be warranted.

For the cancer chemotherapy patient who refuses to consult a neuropsychiatrist, the oncologist can obtain a Minnesota Multiphasic Personality Inventory (MMPI), a psychometric test that will reveal the patient's basic personality structure as well as the psychological aspects of the patient's illness. The results may encourage the patient to accept consultation with a neuropsychiatrist who can then explain the emotional ramifications that are a part of the patient's illness. A solid patient-therapist bond may generate "therapeutic energy" which the patient can tap into to raise the vomiting threshold.

There are four factors of significance in approaching the N&V problem in patients receiving cytotoxic chemotherapy:

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1. *The patient's emotional apparatus:* A number of cancer chemotherapy patients will begin vomiting as soon as they enter the hospital. Some will begin vomiting when only the catheter has been inserted. Others may vomit merely at the sight of an emesis basin. Those who vomit so readily may be termed "high vomiters."

2. *The emetic potential of the agents:* Although nearly all of these drugs have the potential to cause N&V, there is a higher incidence reported for methotrexate (MTX), cisplatin (Platinol), cyclophosphamide (Cytoxan), dacarbazine (DTIC), doxorubicin (Adriamycin), and mechlorethamine (Mustargen).

3. *The nature of the neoplasm:* Neoplastic disease involving the gastrointestinal tract will present added N&V problems, as will metastases from a primary source to the brain or a primary lesion in the brain.

4. *Drug/Drug interaction* (the effect of the antiemetic drug on the cytotoxic agent): Very little is known about these chemical mixes, but the possibility for interaction should be kept in mind. As a corollary, there may be antiemetic drug/drug interactions. One antiemetic combination to be avoided is the concurrent use of metoclopramide (Reglan), with its strong dopamine antagonistic action, and any of the antidopaminergic drugs.

Even should the neuropsychiatrist fail to benefit the patient through psychotherapy, s/he may still effectively assist as psychopharmacologist. Psychopharamcology as a branch of neuropsychiatry had its beginnings in France in 1952. It has grown during the past 30 years to the point where we may now see the beginnings of a new field, one linking oncology with psychopharmacology: oncopsychopharmacology.

A review of the list of drugs commonly utilized for treatment of N&V in chemotherapy patients — prochlorperazine (Compazine), dimenhydrinate (Dramamine), promethazine (Phenergan), trimethobenzamide (Tigan), and thiethylperazine (Torecan)[1] — helps to explain the less than satisfactory results achieved.

When choosing a phenothiazine of the piperazinylpropyl class as an antiemetic, the selection should be trifluoperazine (Stelazine), perphenazine (Trilafon), or fluphenazine (Prolixin). However, this class of phenothiazines (piperazinylpropyl derivatives) is associated with more extrapyramidal side effects (EPS), such as akathisia, than the other important class (dimethylaminopropyl derivatives). Short-term EPS, which includes the long-term complication of tardive dyskinesia, seldom looms as a problem to the neuropsychiatrist, since there is a wide enough selection of drugs to control it. However, of greater importance, the piperazinylpropyl phenothiazines, when used alone, produce little or no sedation in low doses — a disadvantage, for sedation is one of the keys to cancer chemotherapy anti-N&V success. It may, in fact, be the master key. *Table I* shows drug groupings.

Greater benefits can be realized with the phenothiazines of dimethylaminopropyl class. Included are chlorpromazine (Thorazine) and trifluprомazine (Vesprin), two very sedating drugs. Chlorpromazine (CPZ), the forerunner of the antipsychotic drugs, is now only of historical interest to neuropsychiatrists in the treatment of psychoses, having been replaced by second and third generation drugs. Nevertheless, CPZ has several applications in general medicine: It is effective in the treatment of intractable hiccups, especially if used intravenously; it has a role in gastroenteritis of bacterial or viral origin, where it is most effective in the control of nausea and vomiting; and it is ideal — perhaps the drug of choice — for resistant or refractory N&V associated with cancer chemotherapy. CPZ is also the drug of choice in controlling the convulsions of tetanus. Cholestatic jaundice is a possible side effect of CPZ, but is rarely seen at present. In all applications, only small doses — 12.5, 25.0, or 50.0 mg — are indicated. It can be administered orally, intramuscularly or intravenously, and can be repeated as necessary or at fixed intervals. For very resistant cases of N&V, larger doses may be administered without harmful effects.

In the same phenothiazine class as CPZ is trifluprомazine, a much neglected drug for control of N&V. It is conveniently available in tablet and suspension form and also for intravenous or intramuscular use. For the physician hesitant to use CPZ, trifluprомazine is an acceptable alternative.

Promethazine is another widely used antiemetic drug. While also a member of the phenothiazines, it is a dimethylaminoisopropyl derivative, not a dimethylaminopropyl compound as are CPZ and trifluprомazine. Its antipsychotic activity is marginal at best, but it has pronounced antihistaminic activity, which also makes it useful as a sedative-hypnotic. (Straightening the hydrocarbon chain in the molecule yields CPZ, with its major antipsychotic activity.) Promethazine is more effective for postoperative nausea and motion sickness, and far less effective in the control of vomiting associated with cancer chemotherapeutic agents, toxins, and radiation sickness. Rarely it may be prescribed for the schizophrenic patient who complains of insomnia and who may respond to it as an hypnotic.

TABLE I
ANTINAUSEANTS/ANTIEMETICS*

I. Anticholinergic antiemetics
scopolamine
II. Antihistaminic antiemetics
promethazine (Phenergan)
diphenhydramine (Benadryl)
dimenhydrinate (Dramamine)
Bendectin = Decapryn (doxylamine succinate) + pyridoxine
hydroxyzine (Vistaril, Atarax)
III. Antidopaminergic antiemetics
A. Phenothiazines
1. Dimethylaminopropyl derivatives
chlorpromazine (Thorazine)
trifluprornazine (Vesprin)
2. Piperazinylpropyl derivatives
trifluoperazine (Stelazine)
fluphenazine (Prolixin HCl)
perphenazine (Trilafon)
prochlorperazine (Compazine)
thiethylperazine (Torecan)
B. Butyrophenones
haloperidol (Haldol)
droperidol (Inapsine, Inovar)
IV. Sedative antiemetics
phenobarbital
V. Miscellaneous
methylprednisolone (Solu-Medrol)
trimethobenzamide (Tigan)
marijuana (including THC and its semi-synthetic congeners)

* Includes only those drugs mentioned in this paper.

CPZ and trifluprornazine, at one level, act by inhibiting stimuli at the chemoreceptor trigger zone (CTZ). This is true of all antidopaminergic antiemetics listed in *Table I*. A very potent antidopaminergic antiemetic is the nonphenothiazine drug haloperidol (Haldol), which belongs to the butyrophenones. Haloperidol in very small doses — *e.g.* 1-2 mg orally or intramuscularly — is a potent antiemetic for the N&V not only of cytotoxic drugs but also for that caused by radiation. Although the emphasis herein has been on drug-induced N&V, many of the antiemetics mentioned can be similarly utilized to combat the N&V induced by radiation. Unlike CPZ-class drugs, haloperidol is non-sedating. However, EPS frequently occurs with this agent.

A drug of pivotal importance in the control of N&V is the antihistaminic diphenhydramine (Benadryl) which, as a cancer chemotherapy antiemetic, is far superior to its chlorotheophyllinate salt, Dramamine, widely used as an anti-motion sickness agent.

As F. H. Myers *et al.*[2] have noted, there is little differentiation among the antipsychotic-antihistaminic-parasympatholytic group of drugs. Diphenhydramine is invariably classified as an antihistaminic, since that is its best known use, but it also has atropine-like action — i.e., it is parasympatholytic — and it has a pronounced sedative effect. Of special note, it is as effective as trihexyphenidyl (Artane) and benztropine (Cogentin) in the prevention of termination of drug-induced EPS.

Possibly the most effective approach to the treatment of cancer chemotherapy induced N&V is the combination of haloperidol and diphenhydramine. Haloperidol depresses the chemoreceptor trigger zone; diphenhydramine, through its atropine effect, depresses the vomiting center, and with its sedative action it effectively blocks input from the limbic-diencephalic pathways. Diphenhydramine also aborts any possibility of haloperidol-induced EPS. Thus, all recognized avenues for the expression of vomiting are closed off.

There is no paradigm for determination of a regimen of antiemetic drug or drugs for a given patient; the clinician must evaluate each case individually. Each patient's case is unique, yet reminiscent of all others. The choice of drugs and dosages vary dramatically from patient to patient. Some patients may respond to a course of Bendectin (doxylamine + pyridoxine) with dosage adjustments made as necessary; for others, CPZ or trifluprornazine may be effective. If a phenothiazine or the butyrophenone haloperidol fails to prevent emesis when cisplatin or cyclophosphamide is being used, the physician may add diphenhydramine to the regimen. When metoclopramide (the drug of choice in the control of N&V associated with cisplatin) fails, even at the high dosage levels recommended by Gralla *et al.*,[3] large doses of diphenhydramine may be effective. Its sedative action alone may produce the desired result. The skillful psychopharmacologist is not afraid to increase drug dosages. Extensive experience gained in utilizing massive dosages of CPZ for the treatment of schizophrenic patients imparts the necessary sense of confidence to the psychopharmacologist who is called on to treat N&V resulting from chemotherapy.

There are other ways in which the (onco)psychopharmacologist consultant can apply his/her expertise. A young adult with metastatic testicular carcinoma was scheduled to receive combination drug therapy consisting of cisplatin (Platinol), bleomycin sulfate (Blenoxane), and vinblastine sulfate (Velban). Angry at being afflicted with the disease in the first place — the "why me?" syndrome — and

ready to abandon treatment altogether, he approached consultation with a neuropsychiatrist testily and with condescension. A brilliant and successful chemist, he would not willingly have consulted a "shrink." During the discussion of his proposed drug regimen, cisplatin was referred to by the author by its more formal name, cis-diamminedichloroplatinum. The statement that "Platinum, as a heavy metal complex, came into medicine through someone's serendipitous observation that bacteria won't grow on a platinum electrode," interested him enough to stimulate a dialogue.

The patient's knowledge of nutrition had proved helpful to him. Beginning with the diagnosis of cancer, he had become insomniac, and had found his own solution to that problem by taking tryptophan, 1 gm, at bedtime, noting that it was a nutritional substance rather than a drug. On being advised that tryptophan is known to reverse the antitumor effect of Velban, he promised to give up his surreptitious use of tryptophan. The process of developing a patient-therapist bond had begun.

Eventually, he completed the protracted treatment schedule, his N&V controlled at a tolerable minimum level with large doses of metoclopramide. Had metoclopramide failed, large doses of diphenhydramine could have been substituted; a little haloperidol could also have been added, if necessary.

For the patient with life-threatening cancer, it is unrealistic to expect to come to the hospital in the morning for medication and head for the tennis court in the afternoon. With this premise, there is theoretically no reason for anyone to discontinue a course of cancer chemotherapy because of N&V, what with all the sedating antiemetics at our disposal which can be augmented (additively or possibly synergistically) with a second agent of a non-sedating nature.

There are other agents, such as droperidol (Inapsine, Inovar) and methylprednisolone (Solu-Medrol), which have been used with some success to combat N&V of cytotoxic drugs. Lee[4] thinks that methylprednisolone is a potent addition to the anti-N&V drug arsenal, even when the chemotherapeutic agents involved are among those that produce the greatest degree of vomiting.

Although the haloperidol/diphenhydramine combination may be the regimen of choice, hydroxyzine (Vistaril, Atarax) could substitute satisfactorily for diphenhydramine. The medical profession currently is not fully utilizing all available drugs. Phenobarbital, through its sedation, is known to produce an antiemetic effect, and was until recently widely used in the treatment of nausea and vomiting of pregnancy. The parasympatholytic drug scopolamine (L-hyoscine) has ten times the potency of atropine (DL-hyoscyamine) in terms of its effect on the central nervous system — the L-isomer is more potent as a rule than a racemic mixture — and these two drugs are closely related structurally. Scopolamine's application in the N&V problem has been directed toward motion sickness, but it can have a broader antiemetic application if used as part of a combination. Physicians must employ their clinical acumen in mixing parasympatholytics, antihistaminics, phenothiazines/butyrophenones, and even anxiolytics in order to achieve new levels of antiemetic effectiveness.

The use of cannabis as an antiemetic is experimental and of dubious value. Clinical use of marijuana poses health hazards in addition to dangers to DNA, RNA, and protein synthesis in cellular systems.[5] These dangers are corroborated by indepth studies of cannabis as described in the symposia publications of Nahas and colleagues.[6, 7]

Frytak and Moertel[8] have shown that older patients, who clearly represent the largest group of patients treated with cytotoxic medication, respond less than satisfactorily to tetrahydrocannabinol (THC), the psychoactive agent in *Cannabis sativa,* the hemp plant, and suffer a high incidence of undesirable side effects such as hallucinations, tachycardia, and convulsions.

Those who prefer the reefer to the 2.5 and 5 mg capsules currently available under specified regulations, should be aware — as Kagen[9] has shown — that most marijuana contains *Aspergillus fumigatus,* and since cytotoxic drugs lead to immunosuppression, the marijuana-smoking patient in this situation may be inviting an invasive systemic mycosis. However, for the young adult with a history of marijuana use who refuses to try any other suggested approach, THC capsules may represent a solution to the dilemma. The first duty of the physician, after all, is to the patient's well-being, and until better approaches to cancer chemotherapy induced N&V are established, THC can serve the needs of a discrete population of cancer sufferers.

When it comes to THC congeners such as nabilone, the evidence presented by its proponents stands as its own indictment.[10] Whether other semisynthetic derivatives of THC, such as levonantradol, nabitan and naboctate,[11] will prove effective, only time will tell. We have witnessed nearly two decades of widespread interest in marijuana as a recreational drug in our society, but it is only in the past few years that the pharmaceutical industry has turned its attention to the task of trying to find something of genuine

(Continued on page 39)

Refractory Hypercalcemia

Management with Intravenous Fluids and Furosemide Guided by Swan-Ganz Monitoring

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HYPERCALCEMIA associated with malignant disorders remains a difficult clinical management problem. In spite of new and effective pharmacologic alternatives,[1-6] the cornerstone of therapy for these hypercalcemic patients continues to be intravenous administration of saline and furosemide. If appropriately utilized, this method of therapy can be successful, and is much less toxic than some of the newer therapeutic alternatives.[1, 2, 7] A firm grasp of several basic physiologic principles is required, however, to obtain maximum results from the use of saline and furosemide.

Described here is a patient with pernicious hypercalcemia of malignancy whose management illustrates this point. Successful treatment of his hypercalcemia was achieved with the aid of Swan-Ganz catheter monitoring.

Case Report

A 62-year-old male presented to the Kansas City Veteran's Hospital with symptoms consistent with transient cerebral ischemic attacks. A right superficial temporal to right middle cerebral artery bypass graft was performed after cerebral arteriography demonstrated complete right internal carotid occlusion. One week post-operatively, the patient became lethargic and confused. Serum calcium level was 16.7 mg/dl.

Initial therapy included intravenous isotonic saline (5 liters/day) and furosemide (160 mg intravenously daily) for three days. Despite urine production of nearly 5 liters/day the patient's serum calcium level remained unchanged. Subsequently, therapeutic trials with mithramycin, calcitonin, etidronate, indomethacin, and corticosteroids all proved ineffective in reducing the serum calcium level. After ten days, the patient had become comatose, and the serum calcium level had risen to 18.9 mg/dl. At this time a Swan-Ganz catheter was placed to guide vigorous intravenous fluid administration. The relationship between the amount of intravenous fluid administration and urine volume, serum calcium level, and pulmonary capillary wedge pressure (PCWP) during subsequent days is shown in *Figure 1*. As illustrated, extraordinary volumes of saline administration (39.5 liters on day 13) were required to expand the patient's intravascular volume sufficiently to raise the PCWP to 18 mmHg. Furosemide was given only after volume expansion had been accomplished (40 mg twice on day 13). With this therapy the serum calcium level fell to 9.3 mg/dl by day 14, and the patient's mental status improved markedly. During the remainder of the patient's hospitalization until his death, attempts to decrease the massive fluid administration resulted in volume depletion and concomitant symptomatic hypercalcemia (day 15, for example). It should be noted that despite massive fluid replacement the patient did not develop peripheral or pulmonary edema.

A patient with pernicious hypercalcemia refractory to multiple pharmacologic therapies was successfully treated with massive intravenous fluids and furosemide. Management was guided by Swan-Ganz monitoring and careful application of physiologic principles.

Biopsy of a skull lesion revealed metastatic squamous cell carcinoma. Computerized tomography of the chest, abdomen, and head failed to identify a primary source for the carcinoma. Autopsy findings revealed a small primary squamous cell carcinoma of the right lung.

Discussion

The kidney plays a major role in serum calcium homeostasis in both eucalcemic and hypercalcemic states. Ionized and complexed serum calcium are freely filtered across the renal glomerulus and are then reabsorbed along all segments of the renal

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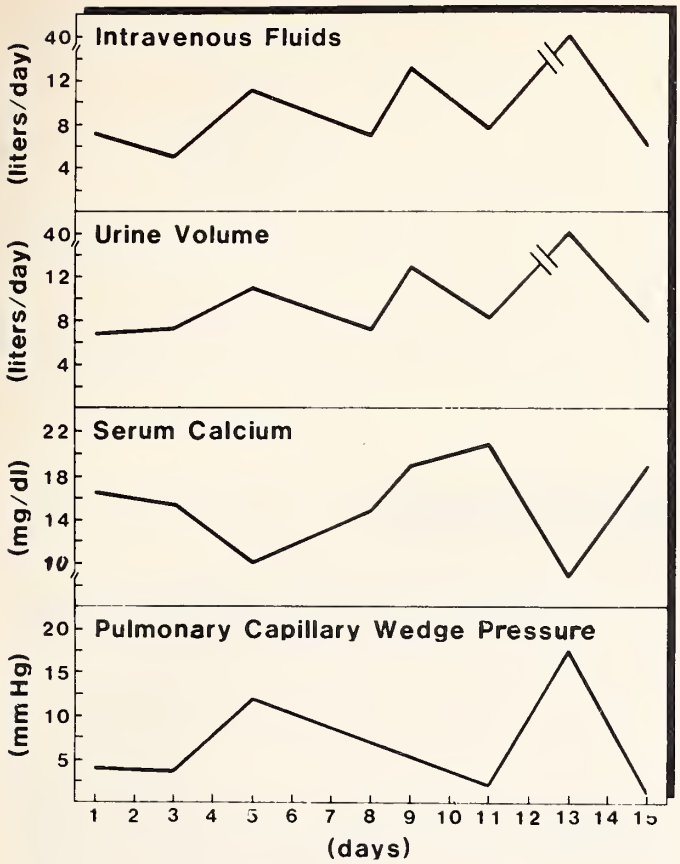


Figure 1. Serum calcium levels, amounts of intravenous fluid administration, urine volumes, and pulmonary capillary wedge pressures (PCWP) are plotted during the patient's hospitalization. Serum calcium was inversely related to the PCWP, reflecting the interrelationship between serum calcium and plasma volume as reflected by the Swan-Ganz PCWP recordings. Note the discordances between the fall in serum calcium and increases in fluid administration and urine volume. Only when fluid administration resulted in plasma volume expansion, as documented by increased PCWP measurements, did the serum calcium level decrease.

nephron.[1, 2] Under normal conditions, about 98 per cent of the filtered load of calcium is reabsorbed by the renal tubules.[8, 9] From 60-80 per cent of net calcium reabsorption occurs in the proximal tubule. Here, calcium reclamation is highly dependent on the effective extracellular fluid (ECF) volume status of the patient and is directly proportional to sodium reabsorption.[1, 8]

Elevated serum calcium levels diminish the renal response to antidiuretic hormone, and thereby lead to renal water loss causing varying degrees of dehydration. This leads to ECF volume contraction which is a predominant feature in most hypercalcemic patients. This situation results in increased proximal tubular calcium reabsorption which then aggravates the coexistent hypercalcemia.

Furosemide acts primarily to inhibit sodium and chloride reabsorption in the ascending limb of the loop of Henle.[7, 10-12] The reabsorption of calcium and magnesium in this segment of the nephron is inhibited by approximately the same percentage as that of sodium.[10, 11] However, because furosemide enhances urinary salt and water losses, and thereby decreases ECF volume,[13] its administration is frequently associated with increased proximal tubular reabsorption of calcium. Decreased calcium reabsorption in the loop of Henle induced by furosemide can be counteracted by increased proximal tubular calcium reabsorption secondary to this volume depletion. Consequently, prior ECF volume restoration and expansion with concomitant diminution in the amount of proximal tubular calcium reabsorption is essential for successful treatment of hypercalcemia with saline and furosemide.

Unfortunately, furosemide is frequently given to hypercalcemic patients without initial restoration of the ECF volume. Simultaneous administration of saline in this setting will increase urine output but, unless the ECF volume is restored first and kept expanded, enhanced renal calcium excretion and subsequent lowering of the serum calcium level may not result.

Thus, it is necessary to expand the ECF volume prior to giving furosemide to enhance urinary calcium excretion. Volume expansion diminishes proximal tubular calcium reabsorption and increases calciuria. Once volume expansion is attained, blockade of the reabsorption of calcium in the loop of Henle by furosemide then greatly enhances calcium excretion.

The patient described in this report is an extreme example that illustrates several physiologic principals of therapy. In *Figure 1*, serum calcium is shown to be inversely related to the PCWP, reflecting the interrelationship between serum calcium and plasma volume status as reflected by Swan-Ganz PCWP recordings. Large intravenous saline infusions, with resultant large urine outputs, did not necessarily result in effective therapy. Note that on day nine, 13 liters of intravenous saline were given and 13 liters of urine were excreted. Despite these large volumes, the plasma volume was not expanded as reflected by PCWP measurements which averaged only 4-5 mmHg. The serum calcium level rose on this day from 13.0 to 16.7 mg/dl, emphasizing the potential discordance between serum calcium levels and urine volume.

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Transcutaneous Nerve Stimulation

Efficacy of Clinical Applications

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ELECTRICAL STIMULATION is routinely used as a method of pain control in many patient therapy centers. The historical use of electricity for afferent stimulation in the control of pain dates back to the early Greeks and Romans; torpedo fish and organs of electric fish were used as their source of electricity. Transcutaneous electrical nerve stimulation (TENS) is a relatively new form of electrical stimulation that has been found to be effective for such diverse pains as headaches, post operative pain, low back pain, arthritis and other pain, both in chronic and acute cases. The use of TENS provides a viable alternative for many patients who might otherwise have undergone surgery or have been placed on a drug regimen. For some patients, TENS is effective as the sole treatment for pain, whereas for others it may be used in conjunction with heat, cold, biofeedback, exercise, or psychotherapy.

Discussed here is the theory of TENS, its current uses in therapy, its relative effectiveness as compared to a placebo, its contraindications, and in particular its relief of chronic wrist arthritis pain.

Theory

The control of pain by TENS is best explained using the gate control theory, described by Melzack and Wall in 1965.[1] It states that stimulation of the skin evokes nerve potentials that send signals to three trunks: (1) the cells of the substantia gelatinosa; (2) the dorsal column fibers; and (3) the central transmission (T) cells in the dorsal horn. The theory is based upon three propositions:

- that the substantia gelatinosa functions as the "gate," modulating the afferent signals;
- that the afferent signals in the dorsal column help to influence the gate control system; and
- that the T-cells activate the neural systems which are then responsible for perception.[1]

Impulses that travel from the periphery in large myelinated fibers are very effective in producing an initial surge of T-cell activity. These large fibers act as a positive stimulus to the substantia gelatinosa, which in turn provides an inhibitory presynaptic effect on the T-cell with an overall effect of decreas-

Because of the highly subjective nature of pain, it is difficult to accurately assess the degree of effectiveness of transcutaneous nerve stimulation for pain relief. As this therapy becomes better understood, it is expected that better results may be obtainable for the patient with chronic pain.

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Editor's Note: Papers published in the Student Thesis series have been written by fourth year medical students at the University of Kansas School of Medicine. Those selected for publication have been recommended by the UKSM faculty.

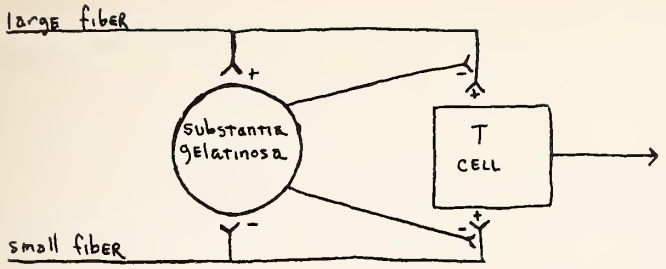


Figure 1. The gate control theory explaining control of pain by TENS utilizes this system. Adapted from Melzack & Wall.

ing the nerve impulses. The small fibers also stimulate the T-cells, but decrease presynaptic inhibition produced by the substantia gelantinosa. This provides an amplification (*i.e.* positive feedback) of the incoming signal. *Figure 1* diagrams this system. Consequently, the overall response of the T-cell is determined by the frequency and activity of the nerve impulses as well as the ratio of small to large fiber activity. Also important is the role that the brain can play in exerting control over sensory input.[1]

In using TENS, electrical stimulation is theorized to produce analgesia by stimulating the large fibers.[2] However, results from experiments inducing nociceptive stimulation in humans have not been able to prove the gate theory as the causative agent in decreasing pain with TENS. A possible explanation is that experimentally induced pain may be different from "acquired" pain. It has been suggested by Fox and Melzack that TENS and acupuncture may work in similar fashions. This would imply that the use of TENS would release opiate-like substances in the periaqueductal gray. Melzack has speculated that the periaqueductal gray region is involved in creating chronic pain. This region is theorized to act as a "reverberating circuit," and TENS may break this circuit, thereby reducing chronic pain.[3]

Others have postulated that TENS may have a local effect by increasing peripheral blood flow. It is undetermined whether these changes in regional blood flow are due to the direct action of TENS on vasomotor fibers or through CNS processing. As can be seen from the number of explanations currently proposed for TENS, there is still considerable doubt about its true mode of action. Empirical data thus far have been the cornerstone for the basis of TENS' use.

Uses

Even though the mechanism of TENS is not understood, it is used in treating a variety of pain, varying from acute to chronic. TENS may be used alone or in conjunction with heat, cold, medications, biofeedback, or any other modality. Types of pain for which TENS has been used include;[4, 5]

arthritis
bone fracture
bursitis
cervical pain
low back pain
muscular injury
causalgia
carpal tunnel syndrome
nerve root injury
peripheral nerve injury
peripheral neuropathy
spinal cord injury
headache
cancer
parturition
phantom limb pain
post surgical pain
psychogenic pain
stump pain
pain of unknown origin
tic douloureux
perineal pain
dental work
thalamic syndrome

TABLE I
DATA FROM LOESER STUDY[4] 1973

Patients with no relief	63 patients or 32%
Patients with initial relief	135 patients or 68%
Patients with long term relief	25 patients or 13%
Patients who stopped treatment	30 patients or 15%

Breakdown of the most common diagnoses:

Description	*Number of Cases*	*No Relief*	*Initial Relief*	*Long term Relief*	*Quit*
Low back pain	61	14	47	8	13
Cervical arthritis	18	3	15	3	1
Extremity arthritis	14	2	12	3	2
Causalgia	13	4	9	2	1
Headaches	13	10	3	1	1

The rest of the categories had fewer than ten patients reported.

TABLE II

EFFECTIVENESS OF TENS — SUBJECTIVE OPINIONS OF PHYSICAL THERAPISTS

Specific use of TENS	*Satisfactory %*	*Partially Satisfactory %*	*Unsatisfactory %*
Acute pain relief	88.5	2.1	9.4
Chronic pain relief	82.8	8.2	9.0
Psychogenic pain relief	50.0	12.5	37.5
Short-term use	95.4	1.8	2.8
Long-term use	68.5	7.8	23.6

The opinions were based on subjective impressions of patient activity, behavior, and stated amounts of pain.

A study performed on 198 patients in 1973 at the University of Washington by Loeser *et al.*[4] provided the data in *Table I*.

A summary of subjective opinions of physical therapists[5] as to the effectiveness of TENS for different types of pain is shown in *Table II*. As before, the best results were with short-term pain patients.

Studies

Pain suppression in areas stimulated by TENS can vary anywhere from a few minutes to seven hours, as found in a study on 100 patients by Picaza *et al.*[6] Ninety per cent of the patients had relief of pain within one hour and many within a period of time less than one-half hour. The time required for pain suppression to appear was reported to be inversely related to the frequency of the stimulation current; low frequencies took longer to produce results. However, low frequency stimulation produced a longer period of anesthesia after termination of TENS.

In some treatments — approximately 10 per cent — the patients reported that the stimulation worsened their pain. The increased pain was often of short duration, but has been reported to last as long as 24 hours. Generally, complaints of pain can be controlled by altering the TENS controls. For instance, decreasing the frequency to a lower rate might alleviate pain.

Stimulation criteria vary for optimal performance with each patient. In Picaza's study,[6] he found constant current (15-40 mA) and constant voltage (20-60 volts) effective. Frequencies of 1-250 pulsations per second (pps) were tested, with the most effective being 25-60 pps. Some patients did experience relief with low frequencies of 1-25 pps. Andersson[7] states that low frequency (2 pps) stimulation was able to decrease pain sensation in teeth up to 300 per cent in healthy volunteers. The effect was long lasting, but when 100 pps stimulation was given, only transitory relief was obtained.

Further work by Andersson on patients with chronic pain produced results virtually contradictory to his study of healthy people.[7] In this experiment, low frequency stimulation yielded poor results with only one patient of 12 experiencing any relief. High frequency stimulation of 100 pps induced pain relief in seven of 12 patients with severe chronic pain. A possible reason for this discrepancy was that patients with chronic pain were not able to tolerate the intensity of stimulation at 2 pps as well as they could at 100 pps. Therefore, at 2 pps, the amplitude was less and consequently, the therapeutic effect was also lessened.

Patients who had positive results from stimulation over a peripheral nerve frequency had relief when remote nerves were stimulated. Similarly, patients who had no pain relief with direct nerve stimulation generally felt no relief with remote stimulation.[6] In Picaza's study, 55 per cent of the patients had relief from pain with TENS therapy. Fifteen per cent of the patients experienced pain relief from remote nerve stimulation, but the amount of relief was generally small. Some patients were helped with subsensory stimulation; this allowed testing of the placebo effect, which will be discussed later. The amount of time the pain was alleviated varied widely from a few minutes to several hours. One patient had complete relief for a period of one week. Conversely, 13 per cent had pain worsening during stimulation, lasting a few minutes afterwards. Five per cent had declining responses with each consecutive therapy session whereas 3 per cent had progressive responses (each session giving longer lasting results than the prior treatment). In addition, it was found that pain elimination was not following the normal skin distribution for a stimulated nerve. Large sections of the body could be treated by stimulation of a single nerve, usually ipsilaterally but sometimes contralaterally. Ulnar nerve stimulation was most

effective for suppression of distant pain. Most often, relief was in the ipsilateral upper extremity, but the contralateral extremity and the lower extremities sometimes experienced relief.

Loeser's study showed that stimulation applied to sensationless regions generally had no results; TENS applied adjacent to these areas has at times given relief from pain. Some patients have even experienced relief when the contralateral area of the body was stimulated. The data from these tests *(Table I)* show a very low long-term gain of only 12.5 per cent, but this value could be artificially low, as 15 per cent of patients who stopped treatment did so for extraneous reasons (*e.g.* attending physician request, or because the insurance company would not cover the treatment). The researchers felt since some patients in this group were showing progress that had they continued, the overall long-term success rate could have been as high as 25 per cent. As such, 12.5 per cent is not a very effective therapy success rate, although it still provides another therapy alternative for the chronic patient.

Other studies[3] have reported good results with TENS. Indeck and Printy documented pain relief in 50 per cent of chronic orthopedic cases and 50 per cent showed improvement from low back pain. Shealy published his work done during a five-year period on 750 patients with 80 per cent of those with acute pain showing improvement and 25 per cent of those with chronic pain experiencing relief. Although this figure is low, 60 per cent of patients with chronic pain had partial but not complete relief.

The use of TENS to control postoperative pain has been studied by Cooperman,[9] who reported on 50 patients who had undergone abdominal surgery. Of those receiving TENS, 35 per cent reported having excellent relief from pain, 42 per cent reported good relief, and 23 per cent reported no relief. The patients who received a sham TENS unit reported only 12 per cent excellent results, 21 per cent good results, and 67 per cent poor results. Therefore, good results may be expected in postoperative cases. These were all subjective assessments of pain by the patient. An interesting finding, possibly related to the theory that TENS works via endogenous morphine-like substances, showed that patients who received narcotics preoperatively had no pain relief from electrical stimulation. No difference was seen in the experimental and control patients regarding pneumonia, ileus, or atelectasis. Also, patients with malignant diseases received less relief from stimulation than those with benign diseases.

Work done by Stratton and Smith[10] showed that patients who had undergone thoracic surgery can have their forced vital capacity increased by using TENS; TENS reduced the postoperative pain allowing the patient to breathe more easily. TENS was also being used to relieve pain from peripheral neuropathy.[11] With the electrical stimulation, the patient acquired complete relief from "burning pain." However, the effectiveness of the relief diminished with continued treatment and to compensate, the patient would increase treatment time. This increasing tolerance to therapy is similar to that of increasing resistance to opiate-containing substances and again may be due to endogenous morphines postulated to be released by TENS.

The best clinical results with TENS are reported at pulse rates of 70-150 pps and at a pulse width of 130 μs.[12] The amplitude of the current should be such that there is a tingling sensation felt by the patient at the site of stimulation, but no pain. The upward rise of the pulse is the most effective in creating an action potential in a nerve. The greater the derivative with respect to time, the more effective the waveform will be. The most common waveforms produced by TENS units are shown in *Figure 2*. No one waveform has been found overall to be better than another, although patients may respond more optimally to one.

For treatment, electrode placement is just as important as the stimulation parameters. Electrodes can be placed in the following locations:

- At "specific points" which correspond to an acupuncture or motor point. Many of the acupuncture sites are over peripheral nerves.
- Over the painful region, without regard to dermatomes or peripheral nerves.
- In the area of the dermatome. When one dermatome covers anterior and posterior areas, dual stimulation may be useful.
- Over the specific spinal cord area that would be supplying the nociceptive input. Electrodes can be

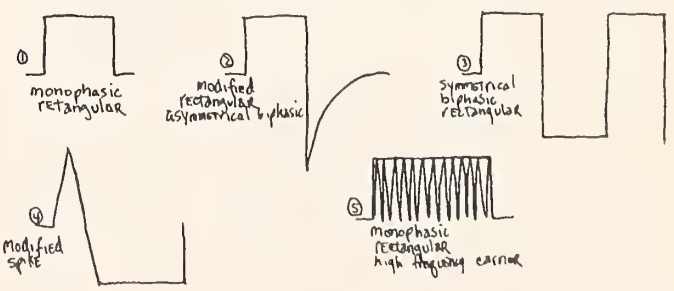


Figure 2. Common waveforms produced by TENS units. Modified from Lampe.

placed next to the vertebrae or between the spinous processes. Spinal cord stimulation in conjunction with dermatomal stimulation with a dual stimulator has been helpful.

- At the place where a peripheral nerve becomes superficial. Stimulation distally to a peripheral nerve lesion may cause pain to worsen because of a blocking effect of the TENS unit itself.
- At a nerve plexus, especially the brachial plexus.

Several of the sites may be used simultaneously for best results. Other placements of electrodes include: (1) dual channel placement so that four electrodes at once may be used; (2) bilateral placement of electrodes for unilateral pain. This is based upon Picaza's findings of some contralateral transfer of relief; (3) contralateral placement when the painful area can not be directly stimulated; (4) enclosing of painful area in a rectangle (or triangle) of stimulating electrodes; or (5) using the stimulator on an unrelated nerve.

By Picaza's study, the ulnar nerve has been the most effective in decreasing pain everywhere except the head. Ulnar stimulation has also been effective for low back pain. This mode of therapy is usually a last resort effort.[13]

Placebo Reaction

The use of TENS as a mode of therapy can be justified only if its results are significantly better than a placebo. In a study by Beecher of 1,082 patients, 35 per cent were helped with chronic pain by an oral placebo.[14] In 13 other clinical double-blind studies, 36 per cent of patients were relieved from pain by a placebo. Surprisingly, the analgesic effect of placebos is proportional (54-56%) to the strength of the supposed medication. Therefore, a placebo could be 56 per cent as powerful as morphine or 56 per cent as powerful as aspirin, depending on which drug was supposedly administered. Two important points of placebo analgesia are: (1) Placebo medication tends to be more effective in patients with severe pain and (2) the effectiveness of the placebo may decrease with its continued use.[14] Nevertheless, the placebo aspect of producing analgesia is definitely important.

A study by Thornsteinnson on 93 patients with chronic pain showed that the placebo effect of TENS was 32 per cent (32% of patients receiving only placebo experienced relief) while 48 per cent of patients had relief with actual stimulation.[15] The placebo effect was proportional to the actual stimulators effect; that is, where the stimulator gave the best pain relief, the placebo also gave the best results. The placebo and stimulator worked best when placed over the center of pain, less well when treating a related nerve trunk, and least well when treating an unrelated nerve trunk. Patients reported that they had longer lasting relief with the stimulator *vs* the placebo, but the difference was not significant. The TENS stimulator and the placebo had about equal adverse effects. In 11 cases, the pain was aggravated by the electrical stimulation and it was worsened, eight cases with placebo.

Rheumatoid Arthritis

The application of TENS to relieve pain in chronic arthritis patients will be specifically addressed, along with some new data collected in a study at the Veterans Administration Hospital, Kansas City, Missouri. Two studies have been performed on patients with severe wrist pain of long duration. Both studies evaluated the pre- and post-treatment pain levels and the overall gain from the treatment.

The first study by Mannheimer, Lund, and Carlsson[16] involved 19 patients. All had wrist rheumatoid arthritis of 1-44 years duration. TENS stimulation was at 70 pps and pulse duration was 0.2 ms. Electrodes were placed over the site of pain (on the dorsal and volar surfaces of the wrist), and three forms of stimulation were used:

1. The amplitude was intense enough to produce paraesthesia in the fingers.
2. The stimulation was weak, so only a slight vibration was noticed.
3. Distant electrode placement was used, with weak stimulation, *i.e.* so the patient felt only a vibratory feeling.

Stimulation was given for five minutes and a weight bearing test evaluated the degree of pain still in the wrist. The weight was chosen so that the patient could not hold it for more than 30 seconds prior to the TENS treatment. The treatment was alternated between wrists and repeated three times, yielding an average value for each wrist.

Eighteen of the 19 patients had increases in weight bearing time after TENS was applied as in stimulation form *number one*. Fourteen had greater than 100 per cent increases, two had greater than a 50 per cent increase, and two had more than a 25 per cent increase. Fourteen of the 19 patients had increased loading time when the TENS stimulation was weak (form *# 2*), and one patient responded with increased loading time with distant electrode placement (form *# 3*). The results are summarized in *Table III*. All measurements of loading time were with the TENS off.

TABLE III
RESULT OF FIRST MANNHEIMER[16] STUDY

Amount of Improvement	*TENS 1*	*TENS 2*	*TENS 3*
100%	14	10	1
50%	2	3	0
25%	2	1	0
None (25%)	1	5	18

Adapted from Mannheimer.

Two patients reported relief of pain in both wrists after just one had been treated; pain was relieved in areas distal to the electrodes exclusively. Four patients with an initial increased loading time were tested six hours later and showed no decrease in abilities. Twenty-four hours later, three patients still had improved functioning. Mannheimer states that this long after-effect time indicates that a stable physiological process was involved, and not just a placebo effect.

The second study was performed again by Mannheimer in nearly an identical fashion.[17] Twenty patients with severe chronic wrist rheumatoid arthritis were tested in the same manner as in his previous study, with pain relief evaluated for different stimulating frequencies. Three forms of stimulation were used:

1. 70 pps stimulation
2. 3 pps stimulation
3. Stimulation at 70 pps with 80 ms duration at a repetition rate of 3 pps.

The stimulation period was ten minutes.

Of the patients who received 70 pps therapy, 90 per cent were able to increase weight bearing time by 100 per cent. Average relief of pain from just ten minutes of stimulation was 18 hours. With 3 pps therapy, only 30 per cent of the patients experienced 100 per cent improvement in loading time, while their relief from pain lasted an average of four hours. The 3-70 pps treatment gave intermediate results. Eighty per cent of the patients were able to increase their loading time by 100 per cent, and the average length of relief from pain was 15 hours. This study is in agreement with Andersson's study, showing that the higher frequency TENS therapy has a better chance of relieving pain. However, in contrast to Picaza's study, the low frequency stimulation had a shorter after-effect period. The length of time the effect lasted in the 70 pps TENS study was extremely long. By comparison, Thorsteinsson[15] reported an average relief time of 3.8 hours after 20 minutes of stimulation. One of Mannheimer's patients reported relief for 72 hours following stimulation. While this certainly is the rare exception, it points to the subjectivity inherent in this type of measurement. Such subjectivity makes comparison between this study and others difficult because these are merely opinions on pain relief. Mannheimer also theorized that the poor results from 3 pps stimulation might have been due to a lower intensity of stimulation used (since the patients' subjective feelings about the stimulation and not a constant current setting were used). An alternate theory was that the different frequency stimulation worked via different mechanisms. *Table IV* outlines these points.

The studies performed at the VA Hospital had a different and simpler experimental design. Two variables were measured on each patient — a weight tolerance and grip strength (as measured by a dynamometer). Stimulation was by a Neuromod dual channel stimulator using only one channel. The frequency was kept at 80 pps because of the generally good results reported at this stimulator rate. Electrodes with a $9cm^2$ area were used in conjunction with electrode paste. The study was performed on just one wrist (the one with the greatest arthritis); the other wrist was left as a control. Because of the reported cross-over effects to the contralateral wrist,

TABLE IV
FINDINGS FROM SECOND MANNHEIMER[17] STUDY

70 pps	Rapid, transient increase of pain threshold	Does not release endorphins	Good effect on pain of disease
3-70 pps	?	Releases endorphins	Good effect on pain of disease
3 pps	Increases the pain threshold	Releases endorphins	Poor effect on pain of disease

Adapted from Mannheimer.

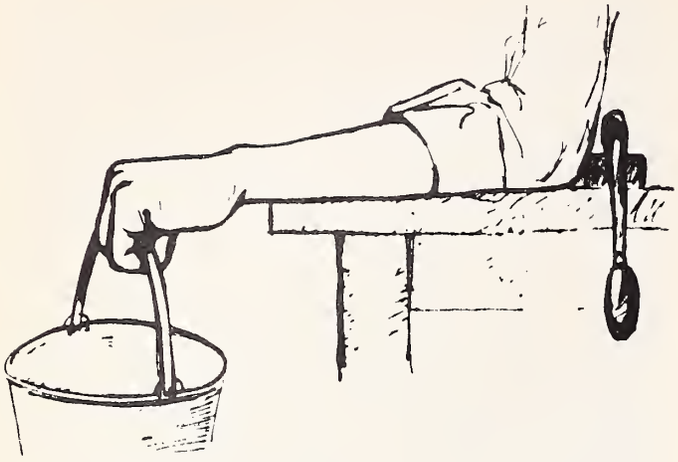


Figure 3. Test for weight bearing capacity. Adapted from Mannheimer.

stimulation of both sides might have resulted in confused data. Also, stimulation time was increased to 15 minutes, since short periods of five minutes may not be long enough to provide adequate effects.

The procedure for the TENS study was:

1. With a straight wrist, the patient was asked to hold a weight in his hand, the weight selected in such a way that the patient could hold it for no more than 30 seconds before experiencing pain in the wrist or before aggravation of existing pain *(Figure 3)*. Each wrist was also tested with a dynamometer to determine the amount of wrist grip. These tests determined the most involved arthritic wrist and set the controls for the treatments.

2a. The placebo effect was first measured by applying the TENS unit to the patient in the normal fashion, telling the patient the unit was on, but the unit was switched off immediately. The patient had his back to the unit so he could not read the controls.

b. The method of using the TENS was as follows: The electrodes were placed on the wrist (one dorsal and one ventral) with electrode paste and then taped in place. The electrodes were then connected to the stimulator and therapy was applied for 15 minutes. In the placebo case, the patient was told the electrical level was low enough so that no stimulation should be felt. In the actual study, the electrical stimulation was set at sub-pain levels, but at a high enough level to elicit some anesthetic effect. The frequency was kept constant for all patients at 80 hz.

3. At the end of the placebo "stimulation," the wrists were loaded with the weight determined in step one, and the length of time of load tolerance measured. The dynamometer recorded any changes in grip strength.
4. Actual TENS stimulus was next applied. As described, TENS amplitude is increased to the point where the patient complains of pain, and is then decreased slightly, so there is some tingling sensation. Stimulus is applied for 15 minutes.
5. After 15 minutes, the two parameters were measured (duration of load tolerance and grip strength) while TENS was still on.
6. Five minutes after TENS was turned off, the two parameters were re-measured.

TABLE V
RESULTS OF STUDY AT VA HOSPITAL, KANSAS CITY MO

		PLACEBO				*TENS*				*REST*			
		Dynamometer		*Weight*		*Dynamometer*		*Weight*		*Dynamometer*		*Weight*	
Patient	*Wrist Stimulated*	*R* %	*L* %	*R* %	*L* %	*R* %	*L* %	*R* %	*L* %	*R* %	*L* %	*R* %	*L* %
A	Right	+ 7	−18	+83	0	− 7	−32	+1120	+ 56	+ 7	−32	+966	+125
B	Left	+13	−19	+33	+100	0	−11	+ 103	+ 83	− 5	+13	+ 84	+162
C	Right	+40	−28	−11	− 29	+40	−64	− 44	+ 7	+40	−14	+ 89	+176
D	Right	0	+ 8	+ 9	− 17	0	−15	+ 9	+ 10	0	+ 7	+ 28	+ 60
E	Left	0	−23	+11	+ 36	− 6	+15	+ 17	+36	+11	0	− 11	+ 27
F	Left	0	+11	+56	+ 98	−20	+22	+ 28	+117	0	+39	+ 19	+143
G	Right	−17	0	+39	− 11	−14	−11	+ 78	− 26	−14	0	+ 5	− 26
H	Right	−13	7	not testable	+115	−13	+ 7	untested	− 8	−13	0	untested	+ 54

Figures show *net* change from pre-treatment values.

At this writing, there have been eight patients seen in this study. *Table V* presents the data collected from the experiment, showing net changes (Δ%) from the pre-treatment values. The percentage changes for the weight bearing trials best represent the treatment's effect on wrist joint pain; the values from the dynamometer best illustrate any changes in grip strength.

There is no general consistency in either tests of grip strength and weight bearing between the measurements made with the TENS unit on and after five minutes of rest with the unit off. For example, between these two measurements, the treatment side may have made positive gains in strength or losses and the control side also made variable changes. The only patients who made definite improvements were A, B and F, although patient F made good improvement with the placebo. Fifty per cent of the patients made gains in weight bearing after the TENS unit was off. In the studies performed by Mannheimer, the measurements were also made with the stimulation off. From opinions expressed by several of the patients, the tingling sensation from the TENS unit impaired the use of the wrist. The majority of the patients described a tingling along the palm, radiating out to the second, third and fourth fingers; occasionally, the sensations would be felt in the thumb. Therefore, perhaps a more definitive measurement might have been taken immediately after the stimulator was turned off, and then five minutes later. At least for these patients, wearing a TENS device while trying to engage in daily activities would not be very satisfactory.

The results of these tests provide data that is too diverse to provide any major conclusions. Basically our study did not show the same improvements in patients that Mannheimer saw; the patients who experienced improvement with the placebo stimulation had more improvement with TENS, and those who had no real benefit from the placebo were not helped with the stimulation. Our method of stimulation (over the wrist area) provided much better improvement in wrist loading time than in grip strength tests. In two patients (C, D) the best improvement was seen on the contralateral side.

The results of these tests might have been better if trials had first been run to determine the best area for stimulation. Also, the way of measuring improvement may give misleading data. Although use of a dynamometer and a weight bearing test provides objective data which can be compared and analyzed, it may not provide any more useful information than a patient's subjective opinion. For example, if patients had exerted themselves on the initial dynamometer test and then felt worn out, subsequent tests would have shown lower readings. This would indicate fatigue, and could easily be confused with the effects of TENS. Future work could include the patient's subjective feelings about the relief of pain and these comments could be compared to the data gathered from the tests.

Contraindications to Therapy

TENS is contraindicated in patients with cardiac pacemakers, and stimulation should not be attempted over the carotid sinus. Electrode jelly may produce a local rash in some patients. Some patients may become "addicted" to the stimulator, but there does not seem to be any physiological dependence or withdrawal reactions.[4]

A study performed by Abram[18] showed ipsilateral temperature changes during stimulation, with the temperature either increasing or decreasing. The TENS unit is capable of stimulating afferent and efferent sympathetic fibers. A patient in their study had a temperature drop on the stimulated hand from 1.5 to 3.3 C. This was postulated to be due to reflex sympathetic hyperactivity. After a stellate ganglion block, this temperature drop upon stimulation did not occur. The patient used TENS at home for some time, but the pain became worse after three months. It was felt that the new radiation of pain may have been due to reflex sympathetic hyperactivity from the past frequent stimulation. It was suggested that skin temperature be monitored initially to detect any autonomic imbalances.

Leyson *et al.*[19] performed a study to determine if there were any detrimental effects on the vesicourethral function of spinal cord injury patients using TENS. Their study involved 17 male patients (nine quadraplegics and eight paraplegics) and gas cystosphincterometry and rectal sphincter electromyograms were performed. Bladder pressures were measured and were shown to increase from 35 to 128 cm H_2O during stimulation; there was also an increase in residual urine volume concommitant with an increase in external sphincter pressure. There was also an increase in rectal sphincter pressure as shown by sphincter electromyograms. These changes occurred when stimulation was applied to dermatomes C5, C6, and T10. No adverse effects on bladder function were seen in patients still in spinal shock who had received TENS for pain relief. It was felt that stimulation over the cervical region may have induced electrical activity in the spinal cord accounting for changes in detrusor and sphincter functions. No significant urethral or detrusor changes were noted for stimulation in the L5 to S2 region.

TABLE VI
SUMMARY OF MAJOR STUDIES

Experimenter	*# of Patients*	*% Experiencing Relief*
Picaza	100	55
Loeser	198	68 initially, 12½ long term
Andersson	12	58
Indeck & Pinty	40	50
Shealy	750	80 for acute cases 25 for chronic cases
Long and Hagfors	400	44
Linzer	23	90 initially, 30 after 3 days
Thornsteinsson	93	48

Summary

Table VI has been compiled from the major studies that have been reported, and presents a review of some of the results achieved with TENS. By 1978, nearly 65,000 cases of TENS therapy had been reported, according to a paper by Ray.[20] Of these cases, 80 per cent reported short term gains, while 35 per cent reported long term gains.

Summarized results of various TENS studies include:

1. TENS can provide relief to some patients for a wide variety of pain.
2. Those who experienced a relief of pain generally felt a tingling sensation from the electrodes, and the electrodes generally perform best when over the painful area.
3. There seems to be a significant placebo effect with TENS.
4. Patients who have no benefits from initial TENS treatment often have no improvement with treatment unless other electrode placements are tried.
5. Patients with multiple areas of pain need multiple channel stimulation.
6. Some patients may have pain worsened by TENS; others may have relief for hours to days after treatment.[21]

Work by Linzer[21] indicates that the best electrical characteristics for therapy are:

1. A minimum current of at least 24 mA; best results were in the ranges of 20-30 mA and 50-60 mA.
2. The best current densities were 3.4-4.5 mA/in^2 and 6.5-7.5 mA/in^2.
3. The best charge per pulse (with 42% of patients experiencing the good results) was at 1.5-2.5 μ A·s.
4. The best pulse width was in the range of 50-100 μs.
5. The best frequency range was between 0 and 60 pps.

While the controversy continues over the way in which TENS works, there is little doubt about its usefulness for relief of pain. Best results require adequate time with the patient to determine the optimum placement for electrodes, and the optimal electrical characteristics for the stimulatory waveform. In the future, it is expected that TENS will be used more frequently as a first means of therapy for the patient with pain. With an increased understanding of this mode of stimulation, perhaps better results may be obtainable for the chronic pain patient. Only continued research and studies will allow this goal to be attained.

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(Continued on page 38)



Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Allogenic Bone Marrow Transplantation for Aplastic Anemia and Acute Leukemia

MAMMO AMARE, M.D.,* *Kansas City, Kansas*

Bone marrow transplantation (BMT) has emerged during the past decade as a useful tool in managing patients with severe aplastic anemia, acute leukemia, and primary immune deficiency states. A number of factors have been responsible for its broadening clinical application. These events include a better understanding of transplantation biology aided by wider application of tissue typing, the introduction of potent immunosuppressive therapy, and improved supportive technology with blood products, antibiotics, and parenteral nutrition. The ensuing discussion will outline the steps involved in BMT, describe its major hazards, and summarize its current status in the treatment of aplastic anemia and acute leukemia.

Donor Selection

The usual donor for marrow transplantation is a sibling matched at the major histocompatibility complex (MHC). In humans, the MHC — known as HLA — has at least three loci which determine the fate of the graft. Two loci — HLA-A and HLA-B — are determined serologically. The third — HLA-D locus — is tested in a mixed leukocyte culture (MLC). Identity at the three loci defines the ideal donor-recipient pair for marrow transplantation. Incompatibility at the ABO locus has no adverse effect and is not a contraindication to the procedure.

Transplantation Technique

Marrow transplantation is technically simple to perform. Under general anesthesia, marrow is harvested mainly from the posterior iliac crests of the donor by multiple needle aspirations and pooled in a container with tissue culture medium and heparin. It is then passed through metal filters, transferred to a bag, and administered to the patient intravenously. Except for local soreness, the donor experiences no ill-effects and the risk of general anesthesia is negligible.

Patient Preparation

The choice of preparatory regimens for marrow transplants depends on the underlying disease. In aplastic anemia, the objective of pretransplant therapy is to induce adequate suppression of the recipient's immune system and to allow engraftment of donor cells. This is commonly accomplished with cyclophosphamide, 50 mg/kg intravenously, administered on four consecutive days. In acute leukemia, the pretransplant treatment, in addition to ablation of the immune system of the recipient, needs to eradicate residual leukemic cells, especially from sanctuary sites. Most schedules include high dose cyclophosphamide, 60 mg/kg intravenously on each of two consecutive days, followed by whole body radiation, administered either in bolus (1000 rads) or in fractionated doses (200 rads/day × 6).

Post-Transplantation Care

The infused marrow usually engrafts and resumes function within two to four weeks. In the meantime, the patient has no functional marrow and is at high risk for infection and bleeding. Hence, proper isolation, antibiotic therapy, support with blood products, and parenteral nutrition is often required. Additionally, to reduce or modify the course of

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graft-*vs*-host disease, all patients receive methotrexate for the first 90 days post-transplant. Once normal marrow function is restored, the major hazards of allogenic BMT are graft rejection, graft-*vs*-host disease (GVHD), interstitial pneumonitis, and leukemic relapse when leukemia was the reason for the transplant.

Graft-*vs*-Host Disease

Recently there has been a significant reduction in the incidence of graft rejection and leukemic relapses. By contrast, GVHD and interstitial pneumonitis continue to be the most frequent and troublesome complications. GVHD occurs in two clinical forms.

Acute GVHD develops soon after transplantation and involves mainly the skin, gut, and liver *(Table I)*. Despite the routine use of prophylactic methotrexate following transplantation, 50 per cent of patients will have evidence of this disease. Although one-half of these patients may have a self-limited course, the remaining one-half develop progressive disease which may be rapidly fatal.

Chronic GVHD emerges several months after transplantation. It is manifested by scleroderma-like lesions, increased susceptibility to infection, and occasional involvement of the gut and liver. Thirty per cent of patients who are alive at six months or longer will develop this complication.

Treatment of established acute and chronic GVHD is unsatisfactory. The acute form occasionally responds to prednisone and anti-thymocyte globulin (ATG). The skin lesions of chronic GVHD may be arrested and rarely reversed with a combination of prednisone and immunosuppressive agents. Presently a variety of approaches, including cyclosporin A and in vitro manipulation of donor marrow cells, are being studied and the early results are encouraging.

Interstitial Pneumonitis

Interstitial pneumonitis is a complication closely related to GVHD. Its major features are dry cough, pulmonary infiltrate, and a variable degree of respiratory insufficiency which sometimes leads to pulmonary failure. The cause is uncertain. Opportunistic organisms, including cytomegalovirus, herpes simplex virus, and pneumocystis have been isolated from some patients. The treatment is directed at the opportunistic organism, but in its absence corticosteroids may be tried.

BMT in Aplastic Anemia

Seventy per cent of patients under the age of 50 years who receive marrow transplantation for severe aplastic anemia are cured. *Table II* shows the criteria for severe aplastic anemia. While GVHD remains the major cause of treatment failure, the recent improvement in survival has been due primarily to the fall in the frequency of graft rejection. There is ample evidence that the incidence of graft rejection is directly related to the degree of alloimmunization of the marrow recipient prior to transplant. Thus, patients who undergo transplantation early in the course of the disease, before they have had excessive transfusions, have a better chance of retaining the graft since they are less likely to be alloimmunized. In fact, this strategy of early intervention has virtually eliminated graft rejection, improving survival from 40 per cent in multiply transfused patients to 70 per cent in previously untransfused patients. At our institution, three patients with severe aplastic anemia, under the age of 30 years, have been treated by bone marrow transplantation. Two are alive at 3.5 years and 1.5 years post-transplant. One died of severe GVHD two months after the procedure. All three had multiple transfusions prior to transplantation.

TABLE I
ACUTE GRAFT-VERSUS-HOST DISEASE
CLINICAL PRESENTATIONS

Target Organ	*Clinical Manifestations*
1. Skin	Erythematous and maculopapular rash
2. Gastrointestinal tract	Diarrhea, abdominal pain, malabsorption
3. Liver	Cholestatic jaundice

BMT in Acute Leukemia

About 20 per cent of patients with end-stage acute leukemia unresponsive to conventional chemotherapy, achieve long-term remission. More importantly, when the transplant is done in first remission, 60

TABLE II
CRITERIA FOR SEVERE APLASTIC ANEMIA

Hypoplastic bone marrow (<25% of normal) with at least two of the following:

1. Granulocyte count <500/μl
2. Platelet count <20,000/μl
3. Anemia (Hg <10 gm/100 ml) with corrected reticulocyte count <1%

per cent of patients under the age of 40 years with acute nonlymphocytic leukemia have had sustained complete remission. Hence, early intervention with BMT, before the leukemia becomes resistant to chemotherapy, seems to promote the chance of permanent eradication of the neoplastic clone leading to a long-term survival and possible cure.

Of the 14 patients with end-stage leukemia who received marrow transplantation at the University of Kansas Medical Center, only three survived one year or longer. On the other hand, of the three patients below the age of 30 years, transplanted in first remission, two are alive at 15 months and 2.5 months post-transplant.

Conclusions and Recommendations

In essence, marrow transplantation consists of a transfer of marrow cells from a normal donor to an HLA-identical recipient irrespective of ABO-compatibility. As marrow transplantation is gaining wider acceptance and as patients undergo the treatment early in the course of their disease, graft rejection and leukemic relapse are being gradually eliminated. On the other hand, GVHD and interstitial pneumonitis continue to be the major causes of treatment failure.

Despite its remaining problems, marrow transplantation is now the treatment of choice for patients under the age of 50 years with severe aplastic anemia. Thus, as soon as this diagnosis is established, the patient and family members should have tissue typing to identify HLA-compatible donor(s). If the patient is a suitable candidate, to minimize alloimmunization, the transplant should be done early before the recipient has been exposed to excessive transfusions or has had blood products from family members.

The role of marrow transplantation in the treatment of acute leukemias is expanding. It may be considered the treatment of choice for patients under the age of 40 years, with acute nonlymphocytic leukemia or poor risk acute lymphoblastic leukemia, who are in remission early in the course of their disease, provided a suitable donor is available. Finally, it is still a therapeutic option for refractory leukemias in which 20 per cent of patients may have a long-term survival.

Self-Assessment Questions

1. Incompatibility at the ABO locus between donor and recipient is not a contraindication to marrow transplantation. (T or F)
2. Graft rejection is less frequent in a marrow transplant recipient who has had multiple blood transfusions. (T or F)
3. In a patient with acute non-lymphocytic leukemia, marrow transplantation will result in long-term survival if:
 a. The patient is under the age of 40 years. (T or F)
 b. The transplantation is done in the first remission. (T or F)
4. The main clinical manifestations of acute GVHD are skin rash, diarrhea, and liver dysfunction. (T or F)

(Answers on page 36)

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Hemoccult Screening

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Drug INFORMATION

LINDA HOGAN, M.S., R.Ph., *Editor*

Isotretinoin (Accutane)

Question: Accutane was recently marketed for the treatment of cystic acne. Will you provide general information on the use of this agent?

Answer: Isotretinoin (13-cis-retinoic acid, a vitamin A derivative) was recently marketed in the United States by Roche Laboratories under the trade name Accutane. It is indicated for the treatment of severe recalcitrant cystic acne resistant to conventional therapy, including systemic antibiotics.

Although the exact mechanism of action of isotretinoin is unknown, studies have found it to have an inhibitory effect on sebum production and sebaceous gland secretion.[1, 2] Goldstein *et al.*[2] found isotretinoin (1 mg/kg/day) administered orally to 28 patients reduced baseline sebum production by 60.1 per cent after two weeks of therapy and by 86.4 per cent after eight weeks. Sebum production returned to 51.7 per cent of baseline four weeks after discontinuance of therapy and to 73.8 per cent after eight weeks. This recovery of sebaceous gland activity does not appear to affect clinical efficacy, which continues for months to years. Such a reduction in sebaceous gland activity may be at least partially responsible for the clinical effectiveness of isotretinoin.[2-5]

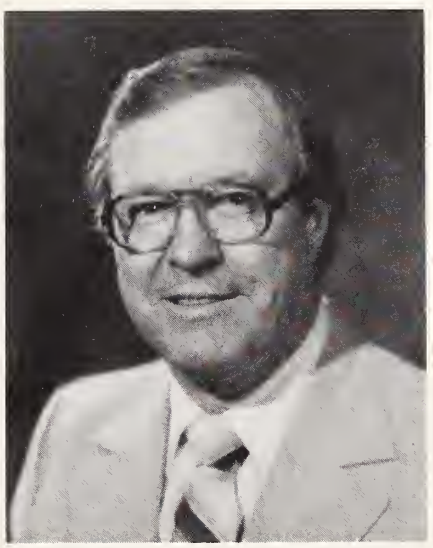
The effectiveness of isotretinoin has been proven by several studies.[2, 3, 6] Peck *et al.*,[6] using a randomized, double-blind crossover study, compared isotretinoin and placebo in the treatment of cystic acne. This study included 33 patients with treatment-resistant cystic and conglobate acne. Of 17 patients in the placebo group, there was an overall 57 per cent increase in the number of cystic lesions. When 16 of 17 patients in the placebo group received treatment with isotretinoin, the number of cysts decreased by 98 per cent. Sixteen patients treated initially with isotretinoin showed a 95 per cent decrease in the number of cysts. Twenty-seven of the 33 patients treated with isotretinoin cleared completely. Eighteen patients received one four-month course of therapy, while 15 patients received two four-month courses. All patients were in remission 38 months after treatment.

Isotretinoin (Accutane) is available as a soft gelatin capsule containing the drug suspended in oil. Administered orally, peak plasma concentrations of isotretinoin are usually obtained within three hours.[7] Following a single 80 mg dose, peak plasma concentrations in 15 male subjects ranged from 180 to 459 ng/ml.[8] Biodegradation of the parent compound in the gut wall and a first-pass effect may contribute to such wide variation in peak plasma concentrations following oral administration. Isotretinoin is at least partly metabolized by glucoronidation and excreted in urine and bile.[8] During chronic dosing, the metabolite — 4-oxo-isotretinoin — accumulates to a ratio of approximately 3.5:1.0 (4-oxo-isotretinoin:isotretinoin). Isotretinoin is 99.9 per cent bound to human plasma (almost exclusively albumin).[7, 8] Its distribution half-life is approximately 0.5 hour while the elimination half-life is 10-20 hours.[8]

Adverse effects of isotretinoin involve the cutaneous and mucous membranes in almost all patients. Clinical and laboratory adverse effects include those shown in *Table I*. Most are dose related and reversible in cystic acne patients with a decrease in dosage or discontinuance of therapy.[7] Patients should be advised to avoid vitamin supplements containing vitamin A due to possible additive toxic effects.

(Continued on page 36)

The President's Message



Dear Colleague:

As the Legislature opens its 1983 session, some issues from the past remain unresolved. One such matter is the issue of nurse practitioners.

The Kansas District Court, in concurrence with the KMS position, recently voided the nurse practitioner law and regulations. In overview, the act was so vague that no one could interpret what a nurse practitioner could do and under what circumstances.

The House of Delegates has twice expressed its support for the concept of allowing nurses privileges and responsibilities concomitant to their training and experience. However, they have clearly stated that nurse practitioners should not be allowed to practice independent of physician review or supervision. KMS efforts to work with the Kansas State Nurses Association to effect a constructive compromise have been unsuccessful. Essentially, we believe the law and regulations should contain language that clearly defines the line of authority for patient care with the physician as supervisor or director of the health care team.

In order to design a new law to establish an equitable system for the delivery of optimum patient care by all members of the health care delivery team, legislators need to understand all aspects of the matter. It is important that you inform your senator and representative of your criteria for legislation that will define future medical practice in Kansas.

We will keep you advised of the status of this matter. In the meantime, your communication with your legislator is essential to our effort to bring about an appropriate resolution to this matter.

President



A Risky Business

We are living in an age of paradox. Life of the common man in its entirety is at least as good as in any previous age and better than most. Even so, there are monumental fears on all sides — fears that are engendered by the very forces designed to allay them. It is a fundamental human characteristic to accept and absorb and thereafter consider as a right each benefit that comes along, transforming it from an event of wonder to the norm. At their inception, though, these benefits impose a degree of risk and danger which provokes a constant feeling of threat by a world that seems to have no interest but to be rid of us. Even the most positive interpretation — the stiff upper lip approach — requires a reminder that all ages have had their dangers and it seems possible that ours appears more threatening largely because we happen to be together at this time. At the negative pole are the perpetual alarmists who would seem to be satisfied with nothing less than removal to a safer world if they knew of one, how to get there safely, and who would qualify as safe companions.

This state of affairs has developed in part because there *is* a constant supply of true risks, history's most persistent repetition and, all in all, one of the more dependable stimulators of new growth. Today's seeming increase appears to be a not unreasonable condition if one keeps in mind the even greater increase in benefits, but there is often a dislocation in time between the incursion of danger and the realization of those benefits so that the relative proportions are not appreciated. To top it off, the media continue, with some justice, to accentuate the negative as being of greater and more saleable news value than the positive, and the certainty of perpetual danger is firmly established.

Three somewhat disparate but related items have brought this to our attention of late. First, at this writing a total heart replacement with an artificial device has been accomplished. The patient is, for the moment, surviving. Whether this continues to be the case or not, the event embodies the risk/benefit dilemma in full measure: risk of life; benefit — minimal perhaps for this patient but, for the experience, better for those of the future who will know improvements of technique.

Second, a recent report calls attention to specific risks relating to fetal surgery which, along with heart surgery, epitomizes those surgical advances (with profound medical assistance) which have within a few decades moved from fantasy to fact. The risks are apparent — to the fetus directly in the process, to the fetus indirectly in sequelae, and to the mother who may be at lesser jeopardy for life or direct effect but still at a level of risk that imposes on the physician a particular obligation of communication and support.

Third — and of a different sort — is a point lifted from an overview statement by F. Gilbert McMahon, chairman of the Commission on the Federal Drug Approval Process. In establishing the premises underlying the process, he notes in part: "Drugs should be acceptably safe in that their benefits should outweigh their risks." No argument there or even surprise (although, it might be noted, without the drama of sophisticated surgery) — but it directs our attention to a vast area of endeavor intimately and continuingly involved with resolving the risk/benefit confrontation.

Even the emphasis on favorable results has the potential for adverse effect as publicity and commercialized promotions create demands for delivery of the miracles faster than reality permits, and have the potential for encouraging the release of the new and insufficiently tried before all potentialities are known — even as the profession's efforts to control the process unintentionally send fearful patients to the charlatans. Given the extent and complexity of progress in all directions, it is, in fact, a credit to the principals that the dangers have not been greater.

Although these considerations could be extended in generality to any discipline, the fundamental na-

(Continued on page 33)

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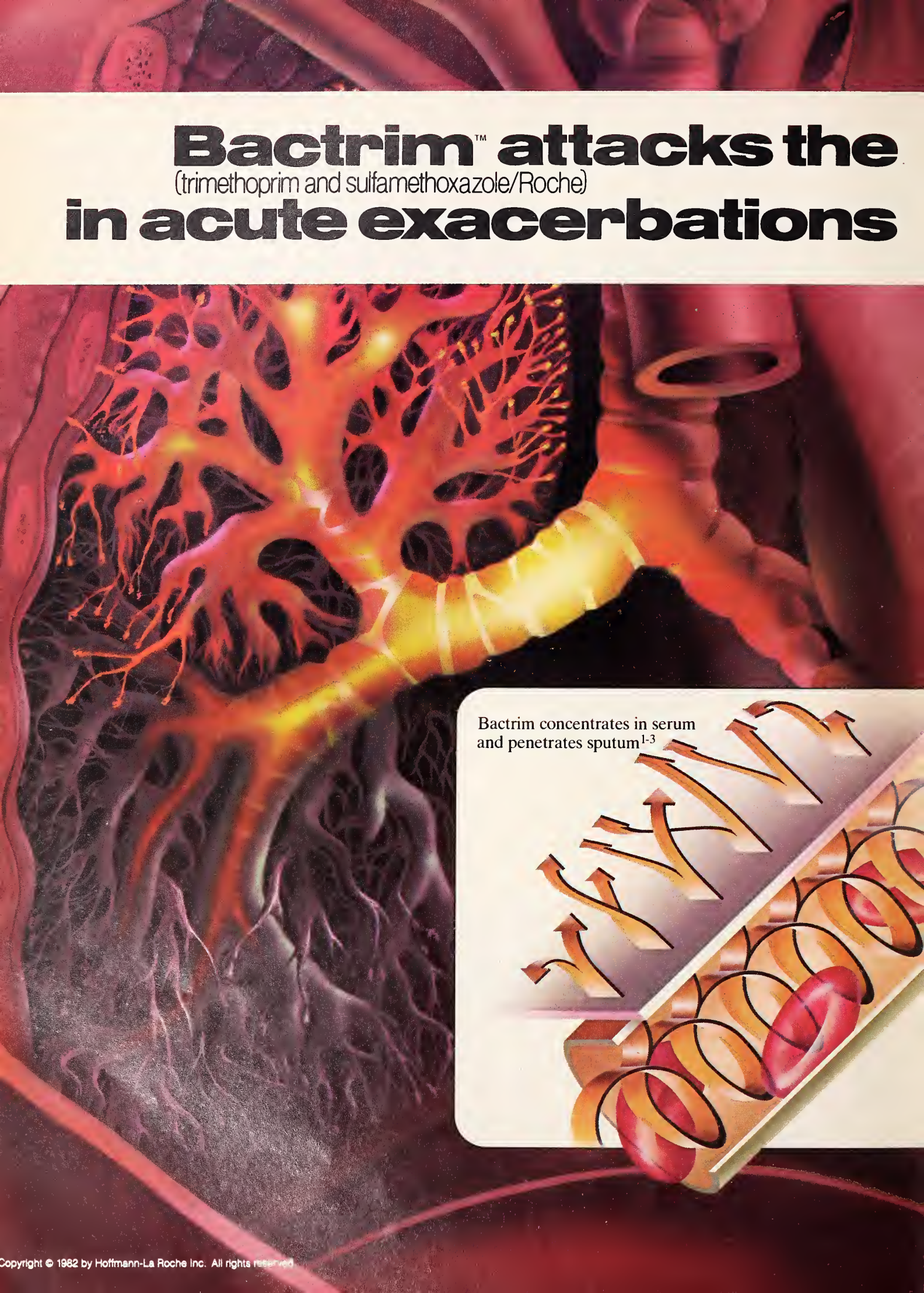
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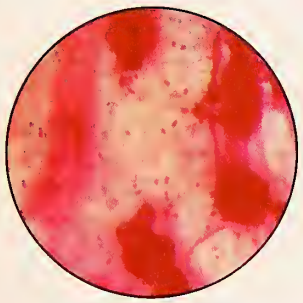
major pathogens of chronic bronchitis*

Bactrim clears sputum of susceptible bacteria

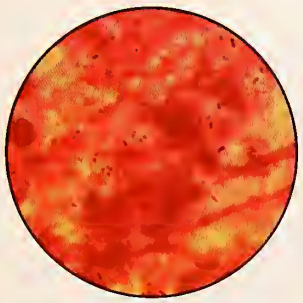
In sputum cultures from patients with acute exacerbations of chronic bronchitis, *H. influenzae* and *S. pneumoniae* are isolated more often than any other pathogens.[4,5] One study of transtracheal aspirates from 76 patients with acute exacerbations found that 80% of the isolates were of these two pathogens.[5]

Bactrim is effective *in vitro* against most strains of both *S. pneumoniae* and *H. influenzae*—even ampicillin-resistant strains. And in acute exacerbations of chronic bronchitis involving these two pathogens, sputum cultures taken seven days after a two-week course of therapy showed that Bactrim eradicated these bacteria in 91% (50 of 55) of the patients treated.[6]

attacks *H. influenzae*—even ampicillin-resistant strains



attacks *S. pneumoniae*



Bactrim reduces coughing and sputum production

In three double-blind comparisons with ampicillin *q.i.d.*, Bactrim DS proved equally effective on all clinical parameters.[7-9] Bactrim reduced the frequency and severity of coughing, reduced the amount of sputum produced and cleared the sputum of purulence.

Bactrim has the added advantages of *b.i.d.* dosage convenience and a lower incidence of diarrhea than with ampicillin, and it is useful in patients allergic to penicillins.

Bactrim also proved more effective than tetracyclines in 10 clinical trials involving nearly 700 patients.[10] Overall clinical condition of the patients, changes in sputum purulence, reduction in sputum volume and microbiological clearance of pathogens—all improved more with Bactrim therapy than with tetracyclines. G.I. side effects occurred in only 7% of patients treated with Bactrim compared with 12% of tetracycline-treated patients. (See Adverse Reactions in summary of product information on next page.)

Bactrim is contraindicated in pregnancy at term and nursing mothers, infants under two months of age, documented megaloblastic anemia due to folate deficiency and hypersensitivity.

Bactrim DS. For acute exacerbations of chronic bronchitis in adults* when it offers an advantage over single-agent antibacterials.

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Bactrim™ DS

(160 mg trimethoprim and 800 mg sulfamethoxazole/Roche)

*Due to susceptible organisms. **Please see next page for summary of product information.**

Bactrim™

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii*. It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis; aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:

Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS:

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

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A Risky Business

(Continued from page 32)

ture of medical practice makes it a prime focus of the risk/benefit relationship and a special significance stems from the fact that these efforts are usually applied to individuals already incapacitated to some extent. It is a largely unappreciated reflection of medical control that the successful resolution of questions of risk, as they apply increasingly to conditions previously considered hopeless, have brought greater emphasis on the quality of life and upon the more mundane problems of living as well. The public has always had an interest in the medical functions but probably never before so much as now or with as much available education in such matters in general and prevention in particular. (It is not to be overlooked, however, that this is not an entirely intellectual exercise. The high price tag on such procedures has probably done more to create pressure for the habit-changing trends in the expectation of future economies.)

A case can be made that the early performance of some dramatic procedure is actually safer than any later ones despite the subsequent experience gained. It is doubtful, for example, that any subsequent candidate for artificial heart implantation will be more carefully selected or indoctrinated, or be more thoroughly informed before consenting. If success is achieved in the early cases, the procedure will be extended in its applications and operators, and the benefits will be then defined by an equilibrium between the stimulus of experience and the restraint of irreducible risks. As for fetal surgery, the same points apply except that a wider variety of applications will be available — and inevitably someone will decide there are dangerous moral or religious transgressions involved. Drug testing, on the other hand, represents a long standing form of risk (even as it is intended to eliminate it) burdened with controls and regulations, and therefore the risk has been greater that potentially valuable agents were denied than that dangerous agents would be released with excessive haste. Even with the strictest scrutiny, some items will produce unfavorable reactions in isolated cases, but if the process of scrutiny seems to have failed in these cases, it at least provides the physician with a reverse benefit in the form of an initial line of defense against possible liability action.

It is not surprising that there has been a revival of the ancient admonition which recurs in the medical teachings of the earliest civilizations: first, do no harm — for the classicists, *primum non nocere*. No principle of medical behavior is rooted more deeply in antiquity or expressed more consistently in various cultures (Hindu, Hebrew, and Greek of definite record), and although a common denominator of medical responsibility exists between those days and the present, the medical format has developed in ways surely unforeseen by those ancient practitioners, giants though they were. Current specifics may not have been in mind but like most statements of sound principle, it remains pertinent to the present scene — although with the customary accommodations which must be made for interpretation in the current light.

Although the durability of the warning owes as much to its ageless generality as to its temporal wisdom, literal application, it can be argued, is impossible since harm may not enter with a fanfare of warning trumpets. Obviously, no physician (outside of fiction or some consumers' advocate's files) would deliberately embark on a harmful procedure for that purpose, whether pursuing an established course or an untried approach. But an obvious element of uncertainty enters when the litigious climate invites the exploitation of every phase of service in detecting some feature that can be classified as harmful. Thus is enunciated the current interpretations: any risk for the patient is a risk for the physician.

Fortunately there is a degree of rationality on the part of the public which accepts, even encourages, new efforts and directions — that is to say, is willing to acknowledge and accept some risks. At the same time, there are enough physicians of creative nature to insist on pursuing new efforts. In spite of misapplications of such efforts or lurid reports of professional malfeasance, medical successes continue substantial enough to provoke public support.

Probably as old as life itself is the inherent understanding that danger is on every hand. Life is, in fact, simply a demonstration that danger has for the moment been allayed. However complex organisms become, the recognition of risks remains within and we are programmed from unremembered existences that danger is *the* fact of life — and if there is any basis for physicians being high in public favor, it is because of their identification with the continuing battle. It is well we devote so much attention and effort to diminishing the dangers to both patient and physician. Considering their prevalence, where would we be if we did not? — *D.E.G.*

Consensus Development Conference

Computed Tomographic Scanning of the Brain

A CONSENSUS Development Conference held recently at the National Institutes of Health reviewed scientific evidence related to computed tomographic scanning (CT) of the brain.

At NIH, consensus conferences bring together research investigators, practicing physicians, representatives of consumer and special interest groups and others to make a scientific assessment of medical technologies and to seek agreement on their safety and effectiveness. After hearing data presented by experts in CT scanning and considering audience discussion, a Consensus Panel of specialists and generalists issued a statement addressing key issues relating to CT scanning of the brain. Following is a summary of that statement:

CT scanning of the brain is a safe, accurate, and powerful tool in the primary diagnosis of brain tumors, brain hemorrhage, major head injury, and certain brain infections. CT scanners have been a major factor in decreasing deaths, especially in severe head injury and brain abscess, and the presence of these conditions clearly calls for the diagnostic assistance of the CT.

While there are numerous indications for use of CT, it should not be employed as a routine screening procedure in cases of minor head trauma or simple headache; its use should be restricted to those cases where structural disease is a reasonable possibility.

Use of CT may be indicated when there is suspicion of arteriovenous malformations, hydrocephalus, herpes simplex encephalitis, parasitic infestations, progressive degenerative diseases of the brain, and intracranial tumors. In primary brain tumors, CT has successfully detected smaller lesions, contributing to lower death rates following surgery and decreased time in the hospital.

CT usually will differentiate between ischemic and hemorrhagic intracranial lesions, enabling the physician to select appropriate medical or surgical therapy. CT is helpful in identifying potential structural causes of complex partial or focal epileptic seizures. It is also an important clinical tool in pediatric neurology that should be used to evaluate undiagnosed coma and other neurological symptoms.

Although CT carries potential hazards — including a slight risk of adverse reactions to contrast material, if used — the procedure is remarkably safe. The amount of radiation produced by CT is comparable to or less than that emitted in the course of other routine diagnostic procedures, including dental x-rays. Physicians should, however, use special care in ordering multiple scans for children because effects of repeated cumulative low-level radiation doses to the immature, developing brain are not yet known.

Although widely used, there are indications that CT may not be sufficiently available for the public to derive the full benefit of its potential. Evidence indicates an insufficient number of the instruments in some large metropolitan areas, in medically underserved areas, and in sparsely populated regions where there is a high incidence of head trauma.

Copies of the full Consensus Statement on CT Scanning of the Brain may be obtained from the Office for Medical Applications of Research, Building 216, National Institutes of Health, Bethesda MD 20205.

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Isotretinoin (Accutane)

(Continued from page 30)

Blood lipid levels should be monitored in patients treated with isotretinoin due to the 25 per cent incidence of hypertriglyceridemia.[7, 9] Pretreatment and weekly to biweekly blood lipid determinations should be obtained until the lipid response to isotretinoin is established.[7]

Isotretinoin is reported to be teratogenic in rats and rabbits and thus is *contraindicated* during pregnancy.[7] Women with childbearing potential should be instructed to use an effective form of contraception during therapy. Contraceptive use should be continued until one month following discontinuation of isotretinoin therapy or until the first normal menstrual flow.[7]

Corneal opacities developed in five of 72 patients treated with isotretinoin, all of whom had normal results in pretreatment ophthalmological examinations.[7] These five patients were treated for a disorder of keratinization; none were cystic acne patients.

The recommended dosage of isotretinoin is 1-2 mg/kg/day administered in two divided daily doses.[7] One mg/kg/day is a normal starting dose which, after a minimum of two weeks, may be titrated upward according to clinical adverse effects and response of the condition.[7] This therapy should be continued for 15-20 weeks or until the total cyst count is decreased by 70 per cent. If necessary, a second 15-20 week course may be administered a minimum of eight weeks after the first.

TABLE I
ADVERSE EFFECTS OF 523 PATIENTS STUDIED

Findings	% *Incidence*
Clinical	
Cheilitis, dry lips	90
Dry mouth	30
Xerosis; desquamation, especially face	30
Pruritis, itching	25
Palmoplantar desquamation	5
Hair thinning	10
Epistaxis, petechiae	25
Conjunctivitis, eye irritation	50
Bone/joint/muscle symptoms	15
Lethargy, fatigue	10
Headache	10
Nausea, vomiting, other gastrointestinal	20
Laboratory	
Elevated sedimentation rates	50
Elevated triglycerides	25
Decreased red cell parameters	10
Decreased white cell counts	10
Increased platelet counts	10
White cells in urine	10
Increases in liver function tests	10

Isotretinoin (Accutane) is available as 10 and 40 mg capsules. The cost to the pharmacist for a 16-week, 60 mg/day course is approximately $381.[10] Thus the cost of this medication should be considered along with its efficacy. Isotretinoin is effective in the treatment of severe cystic acne. Adverse effects are many; thus patients should be monitored closely with dosage adjustment or discontinuation of therapy when necessary. All patients should be aware of the cutaneous as well as other common adverse effects. Obese patients, those with diabetes mellitus, and those with a family history of hypertriglyceridemia may be at increased cardiovascular risk due to the elevation in plasma triglycerides caused by isotretinoin. The teratogenic effects of this agent absolutely preclude its use during pregnancy. Isotretinoin represents a major advance in the treatment of cystic acne. However, clinicians must be aware of the adverse effects and the cost involved. The benefit:risk ratio should be an important factor in a decision concerning its prescription. There is no information available on the use of isotretinoin in less severe acne.

Submitted by
Bruce Scott, R.Ph.
Pharmacy Resident

References are available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

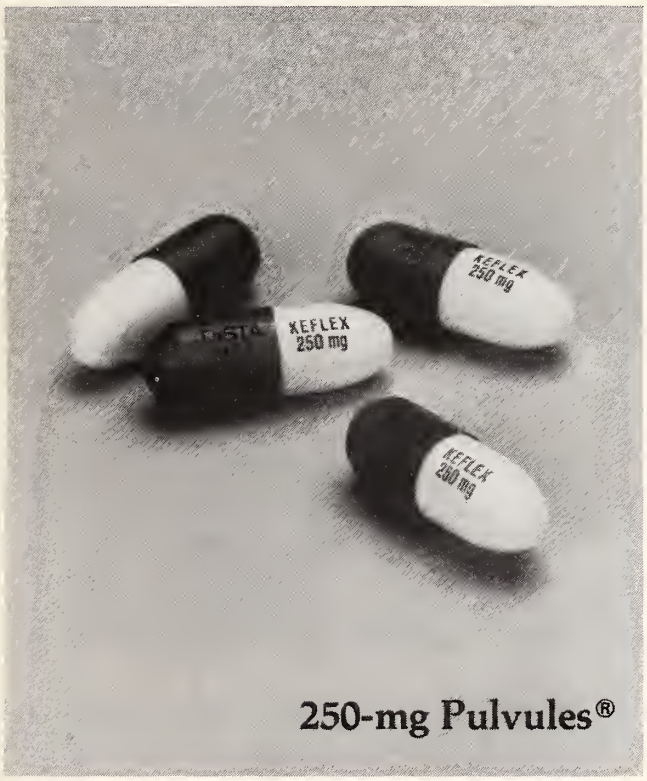
Bone Marrow Transplantation

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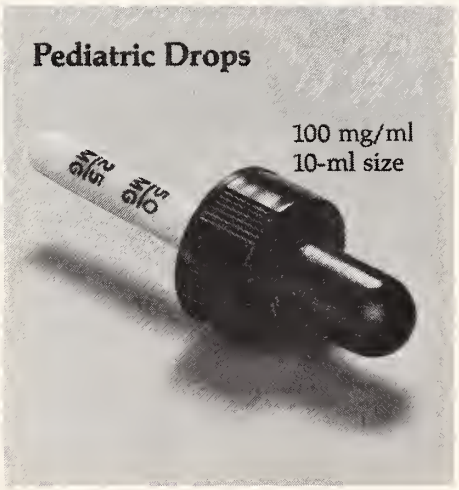
Answers

1. *True*
2. *False*
3a. *True*
3b. *True*
4. *True*

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Hyperbilirubinemia and Mono

(Continued from page 9)

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Transcutaneous Nerve Stimulation

(Continued from page 26)

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Nausea and Vomiting

(Continued from page 15)

therapeutic value (that can meet the legitimate criteria established by the Food and Drug Administration) by working with the cannabinoid moiety. Nevertheless, because of THC's deleterious effects on cellular DNA, RNA, and protein synthesis — properties shared by natural cannabinoids in general — it is doubtful that a congener devoid of long-term hazardous effects can be synthesized;[12] however, that would not rule out the short-term use of such a congener.

Summary

The approach to anti-N&V treatment in the patient undergoing cancer chemotherapy is highly individualized. There is no standard protocol to guide the clinician. It may be a long time before a drug approach is devised that will meet the current double-blind criteria for establishing a new drug's efficacy to the satisfaction of the FDA. The FDA's standards are strictly scientific. What the clinician can offer to his/her patients at present is primarily the art of medicine — and in the field of cancer chemotherapy N&V control, the neuropsychiatrist, of all clinicians, may be in a better position to help.

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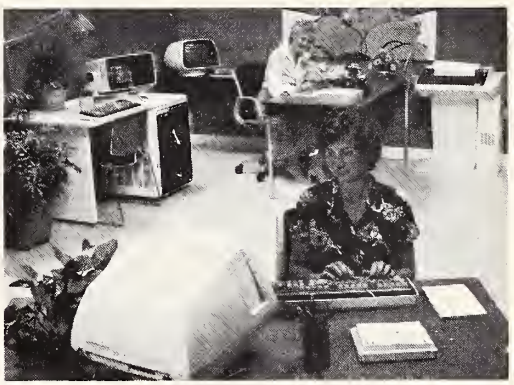
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Refractory Hypercalcemia

(Continued from page 17)

Massive fluid administration of 39 liters of isotonic fluid in 24 hours was necessary to increase the ECF volume and attain PCWP measurements of 18 mmHg (day 13). Furosemide was given twice during this period and only when the PCWP reached 18 mmHg or greater. A 24-hour urinary calcium excretion rate approaching 4.0 grams was achieved with this therapy, and the serum calcium level fell to 9.3 mg/dl. This improvement did not represent simple dilution of the intravascular volume as verified by the fact that the serum hemoglobin and protein levels remained relatively unchanged during this time. Swan-Ganz monitoring was critically important in patient management because of the huge volumes of fluid administered.

While this case represents an exceptional example of hypercalcemia, it does clearly emphasize an often overlooked therapeutic principle. Inappropriate amounts of saline and furosemide are frequently given to hypercalcemic patients without first assuring that the intravascular volume is replenished. Furosemide administration in this setting typically results in increased urine volumes without a concomitant increase in calcium excretion, and presumed failure of this treatment modality. Restoration and expansion of intravascular volume prior to furosemide administration enhances calcium excretion in many of these situations and permits a more judicious use of the more toxic therapeutic alternatives in hypercalcemic patients.

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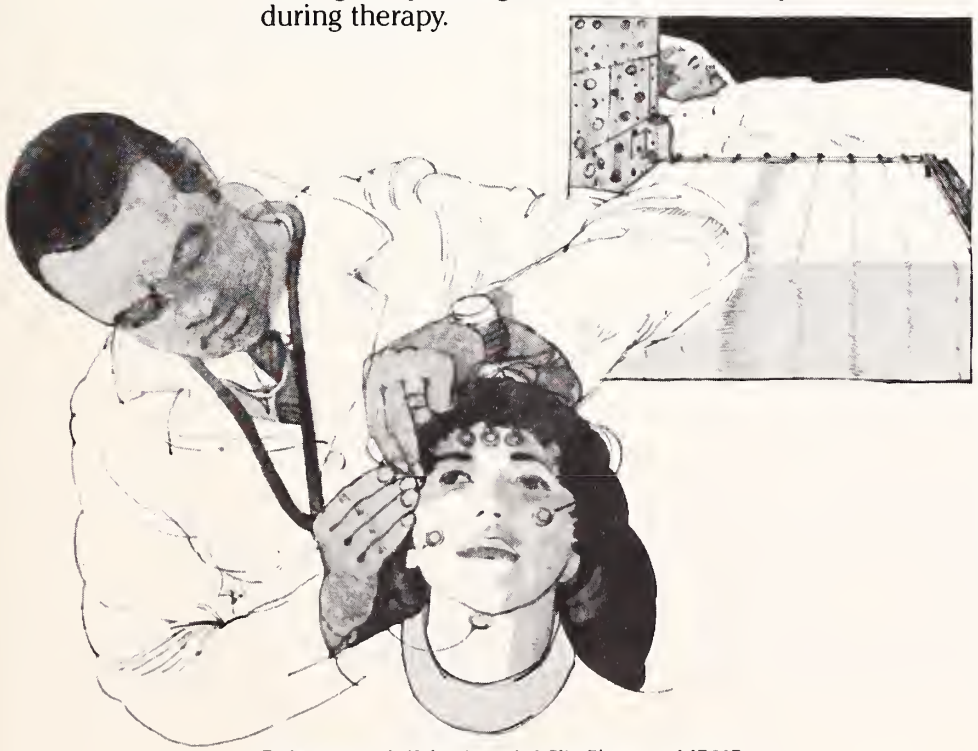
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References: 1. Kales A *et al: J Clin Pharmacol 17*:207-213, Apr 1977 and data on file, Hoffmann-La Roche Inc., Nutley, NJ. **2.** Kales A: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **3.** Zimmerman AM: *Curr Ther Res 13*:18-22, Jan 1971. **4.** Kales A *et al: JAMA 241*:1692-1695, Apr 20, 1979. **5.** Kales A, Scharf MB, Kales JD: *Science 201*:1039-1041, Sep 15, 1978. **6.** Kales A *et al: Clin Pharmacol Ther 19*:576-583, May 1976. **7.** Kales A, Kales JD: *Pharmacol Physicians 4*:1-6, Sep 1970. **8.** Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc 27*:541-546, Dec 1979. **9.** Dement WC *et al: Behav Med 5*:25-31, Oct 1978. **10.** Vogel GW: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **11.** Karacan I, Williams RL, Smith JR: The sleep laboratory in the investigation of sleep and sleep disturbances. Scientific exhibit at the 124th annual meeting of the American Psychiatric Association, Washington, DC, May 3-7, 1971. **12.** Pollak CP, McGregor PA, Weitzman ED: The effects of flurazepam on daytime sleep after acute sleep-wake cycle reversal. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, June 30-July 4, 1975. **13.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ.

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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended. Repeated therapy should only be undertaken with appropriate patient evaluation.

Contraindications: Known hypersensitivity to flurazepam HCl; pregnancy. Benzodiazepines may cause fetal damage when administered during pregnancy. Several studies suggest an increased risk of congenital malformations associated with benzodiazepine use during the first trimester. Warn patients of the potential risks to the fetus should the possibility of becoming pregnant exist while receiving flurazepam. Instruct patient to discontinue drug prior to becoming pregnant. Consider the possibility of pregnancy prior to instituting therapy.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg recommended initially until response is determined.

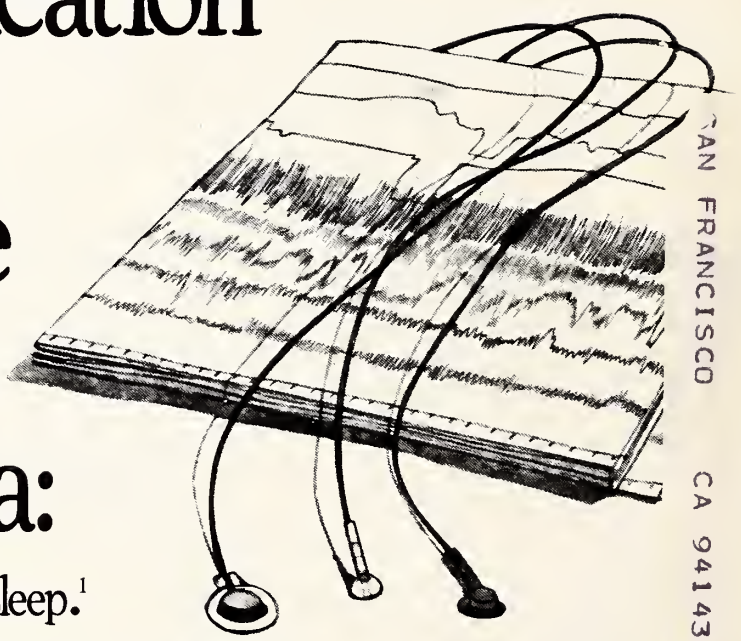
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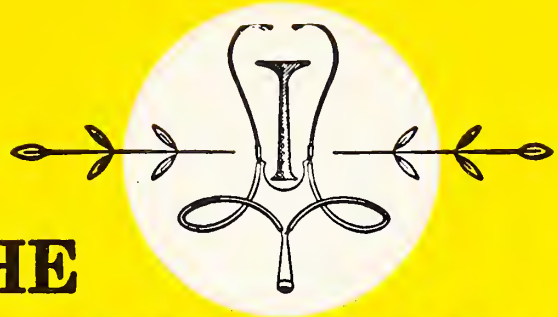
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VOL. LXXXIV
NO. II

AMA House of Delegates

Summary of Actions Taken at the 1982 Interim Meeting, Miami Beach

The 1982 Interim Meeting of the American Medical Association was held December 5-8. Attending from Kansas were Alex C. Scott, M.D., Kermit G. Wedel, M.D., Lew W. Purinton, M.D., and Jimmie A. Gleason, M.D. William J. Reals, M.D., represented the College of American Pathologists.

The House was informed that a preliminary membership count indicates that 25 states will have additional delegate representation in 1983 — the first year that direct members will be applied to the delegate allocation. Kansas is now eligible for a third delegate and alternate.

House Size and Composition

- A report addressing the size and composition of the House generated much discussion. The report was prepared in response to a number of delegate concerns:

— That the votes of state society delegates were being diluted by the number of specialty society delegates;
— That the specialty societies had only one delegate no matter how large or small; and
— That some specialty disciplines were represented by more than one specialty society.

- The House reaffirmed the value of specialty society representation and said that these societies should continue to meet the criteria that gained them a seat in the House.
- The House voted to invite the American Hospital Association and the American Dental Association to send observers to AMA meetings. In the President's Address, Dr. William Y. Rial suggested that the National Medical Association and the American Osteopathic Association also be included. That recommendation was referred to the Board of Trustees for study and a report.
- The House urged state societies to elect medical students and residents to their delegations.
- The House rejected recommendations to provide a delegate seat for state society presidents and to provide proportional representation to specialty societies.

Format and Procedure

The Special Advisory Committee to Study the Annual and Interim Meetings outlined several suggestions on ways to preserve the level of delegate participation while reducing expenses for the AMA and members of the Federation.

- A recommendation that House meetings be shortened by one day was defeated. Delegates were concerned that time would be inadequate to consider House business.
- The House agreed with the committee that the study was worthwhile and voted to formally examine its format and procedures every five years.
- The House reaffirmed its position as the primary policy-making body for the American medical profession and urged its members to recognize a responsibility to represent the AMA throughout the year.

JCAH Manual Revision

The House considered four resolutions pertaining to a proposed revision in the Medical Staff chapter of the JCAH *Accreditation Manual for Hospitals*. After much discussion, the House voted to:

- Commend the AMA Commissioners to the JCAH for their efforts to develop methods of preserving appropriate physician involvement in the maintenance of quality patient care throughout the process of revising the manual; and
- Disseminate the proposed revisions for comment and inform physicians of subsequent revisions through appropriate publications.

The House also voted to reaffirm existing policy on the issue which states:

> RESOLVED, That it is the policy of the American Medical Association that the objective of hospital accreditation is primarily to evaluate the quality of patient care, to provide recommendations for remedying deficiencies and improving the quality of patient care, and to withhold accreditation from those institutions which do not meet an acceptable standard of patient care; and be it further
>
> RESOLVED, That the AMA oppose accreditation requirements which impose rigid, uniform, mandatory administrative procedures, methods of operation, nomenclature, or forms of organization for the hospital, its governing board, attending staff and committees; and be it further
>
> RESOLVED, That the AMA recognize that excellence in patient care is more easily attainable when the accreditation process is flexible and is concerned with evaluating the quality of hospital service and not the administrative procedures, or form of organizations used to provide patient care;

(Continued on page 45)

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The short half-life of Ativan facilitates more rapid response to dosage adjustments, aiding you in titrating therapy to the patient's changing needs. Once you decide to discontinue Ativan, it will be out of your patient's system 4 days after the final dose—unlike the long-acting, multi-metabolite benzodiazepines which take as long as 2 weeks to be totally eliminated.

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References:
1. Klotz U, Reimann I: N Engl J Med *302*:1012-1014, 1980.
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4. Sellers EM, Naranjo CA, Peachey JE: N Engl J Med *305*:1255-1262, 1981.
5. Ruffalo RL, Thompson JF, Segal JL: South Med J *74*:1075-1078, 1981.

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*Pharmacokinetics cannot as yet be directly related to efficacy.
†All benzodiazepines produce additive effects when given with CNS depressants such as barbiturates or alcohol.

See important information on following page.

Ativan®
for (lorazepam) IV
Anxiety

Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan®
for (lorazepam)℞
Anxiety

Dosage: **Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

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Manuscripts must be typewritten, double spaced leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle if frequently used to keep the main title short.

Summary: all manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: the author is responsible for all statments made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

The Journal will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the Journal has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.








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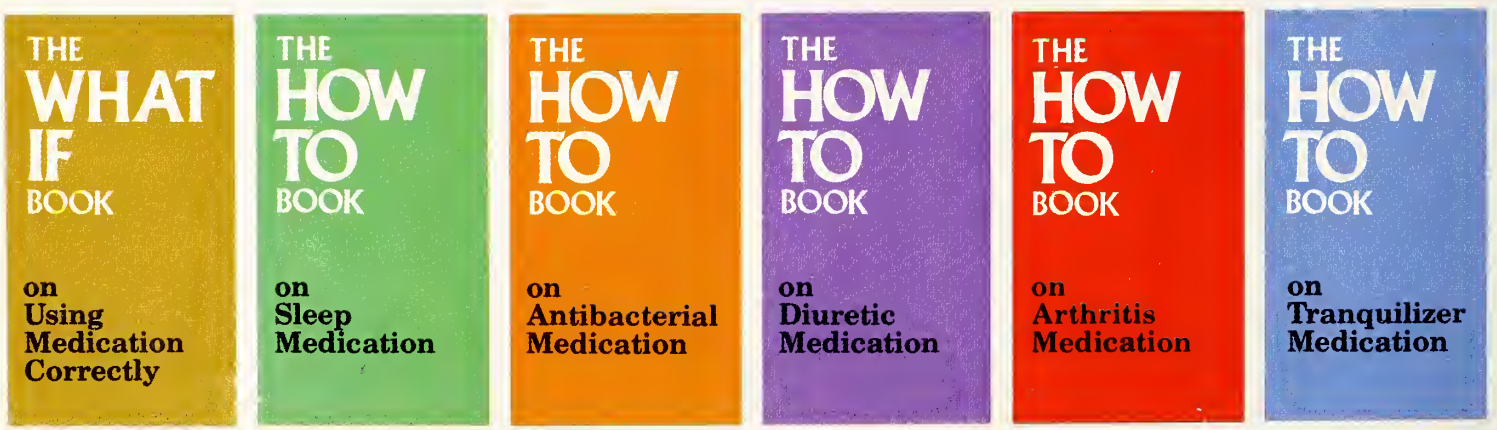
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AMA House of Delegates

(Continued from page 42)

and be it further

RESOLVED, That the Board of Trustees, in its discretion, give the AMA Commissioners to the Joint Commission on Accreditation of Hospitals appropriate guidance for implementing this resolution.

Hospital Medical Staffs

The Study Committee proposed a number of recommendations that together would establish a Hospital Medical Staff Section with a delegate in the House. The House essentially adopted this report with some amendments for clarification.

- Representatives to the Hospital Medical Staffs Section shall be members of the AMA, nominated and elected by the active voting physician members of the medical staff. They must have clinical privileges at the institution they represent.
- Initially representatives to the Section may be selected by the elected physician leadership.
- After July 1, 1984, proof of nomination and election by the members of the active voting physician medical staff shall be required.
- The Section shall be organized as an open assembly to which each hospital medical staff would be eligible to send one elected representative.
- All state and county medical societies are urged to work closely with medical staffs in their areas to facilitate participation in the Section.
- State societies may want to organize a Hospital Medical Staff Section at their annual meetings.
- County societies may want to organize a Hospital Staff Liaison Committee.
- The AMA will offer assistance and support to state and county efforts related to hospital medical staffs.

Dues Exemption

The House adopted new criteria providing for AMA dues exemption to include:

- Members suffering financial hardship and/or disability;
- Retired members aged 65 years; and
- Members aged 70 years regardless of retirement.

All dues exempt members are eligible to receive *American Medical News*, *JAMA*, and the AMA specialty journals at half price.

Peer Review

The House adopted a report that outlined the major provisions of the Peer Review Improvement Act of 1982 and the new peer review program. The report recommended that the AMA:

- Support and work to strengthen those elements of the program that are consistent with present Association policy on medical peer review;
- Seek modification through regulation and amendment of those mandatory elements that are not consistent with existing policy;
- Expand assistance to physicians and medical societies in assuming a leadership role in medical peer review; and
- Also expand assistance in negotiating contracts under the program which would retain professional direction and control with appropriate emphasis on quality rather than cost.

The House also directed that when and if the above efforts are considered unsuccessful, the AMA should seek repeal of the Durenberger bill and report back to the 1983 Annual Meeting.

Competition

The House considered two reports addressing the issue of competition in medicine. One report identified and examined aspects of medicine that are changing in response to competitive economic pressures, including:

- Changes in practice arrangements;
- Relations between physicians and hospitals;
- Reimbursement policies of third-party payers; and
- Professional relations among physicians.

AMA policy supports the concepts of fair market competition and neutrality of public policy among alternative health care delivery systems.

Another report described an AMA action plan designed to help physicians deal with changing market forces. The report identified the following general effects of competition on the practice of medicine:

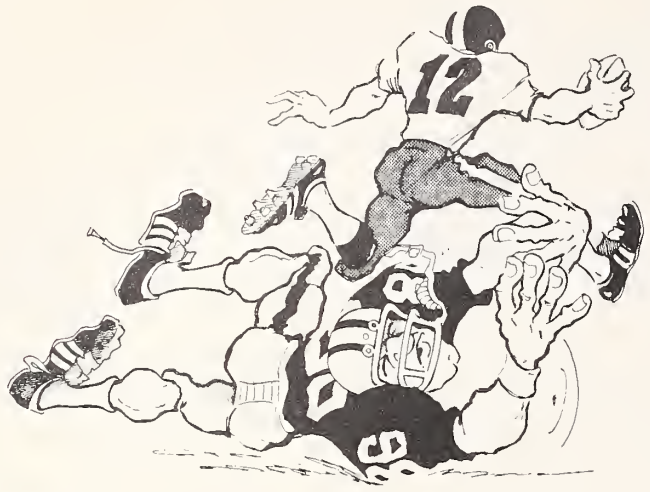
- More aggressive efforts will be made by physicians and other providers to attract patients.
- The nature of practice opportunities will change with respect to location, organization, and specialty.
- The economic impact of competition on physicians will differ with rewards likely to be greatest for those who provide quality care in the least costly manner.

The AMA action plan will help physicians to determine:

- What kind of practice opportunities are available;
- How to organize practice resources effectively; and
- Where to obtain medical practice finances.

Cost Shifting

The House voted to:



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- Continue to oppose changes in the Medicare and Medicaid hospital reimbursement systems that result in cost shifting to private patients; and
- Continue to widely publicize the deleterious effects on the private sector of such cost shifts in efforts to save dollars for the federal programs.

Definition of "Physician"

In response to Resolution 47 that proposed a definition of physician, the House reaffirmed the definition of physician adopted at the 1972 Annual Meeting. The definition reads:

> A physician is a person who, having been regularly admitted to a medical school, duly recognized in the country in which it is located, has successfully completed the prescribed course of studies in medicine and has acquired the requisite qualifications to be legally licensed to practice medicine.

Abuse

The House adopted a Judicial Council opinion on the ethical responsibilities of physicians regarding abuse of children, elderly persons, and others at risk. The opinion states in part:

> The physician who fails to comply with the laws requiring reporting of suspected cases of abuse of children and elderly persons and others at risk can expect that the victims could receive more severe abuse that may result in permanent bodily or brain injury or even death.

The Judicial Council also pointed out that a physician's obligation to comply with statutory requirements is clearly stated in the Principles of Medical Ethics. The ethical obligation of the physician may exceed the statutory limit.

Ethical Guidelines/Accreditation

In another report, the Judicial Council presented ethical guidelines for physicians engaged in accreditation activities. The report states that physicians should apply standards that are relevant, fair, reasonable, and nondiscriminatory and not use accreditation activities for economic regulation.

Alcoholic Beverages

The House voted:

- To reaffirm its position that containers of all alcoholic beverages be required to bear the following statement on their labels:

> Alcohol may be injurious to your health and, if consumed during pregnancy, to the health of unborn children.

- To encourage the media that accept advertising of

alcoholic beverages to refuse such advertising.

Nurse Practitioners

The House adopted the following policy pertaining to Nurse Practitioners:

> That the American Medical Association, in the public interest, urge state medical associations to oppose enactment of legislation to authorize the totally independent practice of medicine by any individual who has not completed the state's requirements for licensure to engage in the practice of medicine and surgery in all of its branches.

TEFRA

The House opposed regulations under the Tax Equity and Fiscal Responsibility Act of 1982 (TEFRA) which would:

- Interfere with and/or redefine the practice of medicine;
- Substitute hourly wages or annual salaries for present reimbursement mechanisms for physicians' service to patients;
- Base physician reimbursement on any system that does not give recognition to knowledge, skill, time, and effort; and
- Otherwise impinge significantly upon the practice of medicine.

The House also directed that such opposition, as required and appropriate, shall be expressed through action in Congress, with the Administration, and in the Courts.

Reimbursement

The House voted that the AMA:

- Reaffirm that payment for medical services be based on the services rendered; and
- Continue its opposition to inappropriate regulations developed to implement the Tax Equity and Fiscal Responsibility Act of 1982 (TEFRA) and take actions appropriate to accomplish the necessary results.

Automobile Restraints/Infants

The House adopted the following policy:

> That the AMA continue to encourage the use of infant and child restraint systems through voluntary education programs and through legislation both at the national level and through the various state societies.

Pertussis Vaccines

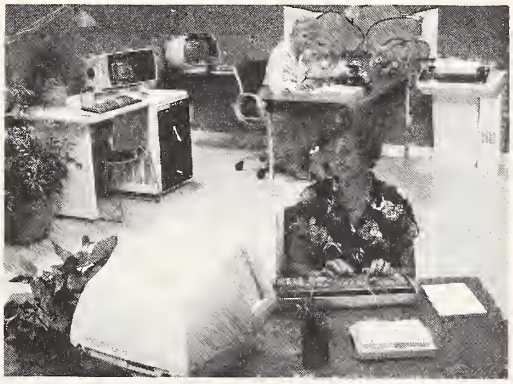
The House adopted the following policy:

> That the AMA support the continued use of per-

(Continued on page 87)

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AUXILIARY NEWS

An Open Letter to Kansas Physicians

Dear Doctor:

Driving the "highways and byways" of Kansas to visit the various county auxiliaries has been a most enjoyable privilege of this office. We have been royally entertained and have enjoyed all the coffees, luncheons, and dinners — as well as the guest rooms in your homes. Last year while returning from a trip to western Kansas, I earned the nickname "The Deerslayer" when my car hit a deer. This year a monstrous bumblebee hitched a ride on some wildflowers picked by my traveling companion, Diane Sanders!

It is evident that KMSA is a strong organization, composed of talented people. Each county is unique in its approach to our goals, and each county has special areas of emphasis in community service.

There are many of you whose spouse is not a member of our Auxiliary. I hope I can convince you of the value of our organization so you will encourage your mate to join. Bringing credit to the medical profession is a fringe benefit of our volunteer activities. It's indirect, but it's real. If the physician spouse takes Auxiliary membership seriously, not only does credit accrue to the medical profession through our service projects, but appropriate information is conveyed during conversation with the public. An uninformed spouse can be detrimental, so we appreciate knowing how to respond to people of the community who get misinformation about the medical profession from the news media. Not the least of reasons for Auxiliary membership is the opportunity it provides to develop friendship with other doctors' spouses. We have seminars geared toward helping members to cope with problems that are unique to this profession.

A benefit of this office is the privilege of sitting on the Board of KMS. The opportunity to participate in the decision making process has been enlightening and interesting.

It is sometimes difficult to be the leader of so many talented, intelligent auxilians — all with good ideas — and I sometimes feel like Charlie Brown when Lucy said, "Well, you win some and you lose some, Charlie Brown." And Charlie Brown replied, "Gee, that would be great!"

Meldon Laury
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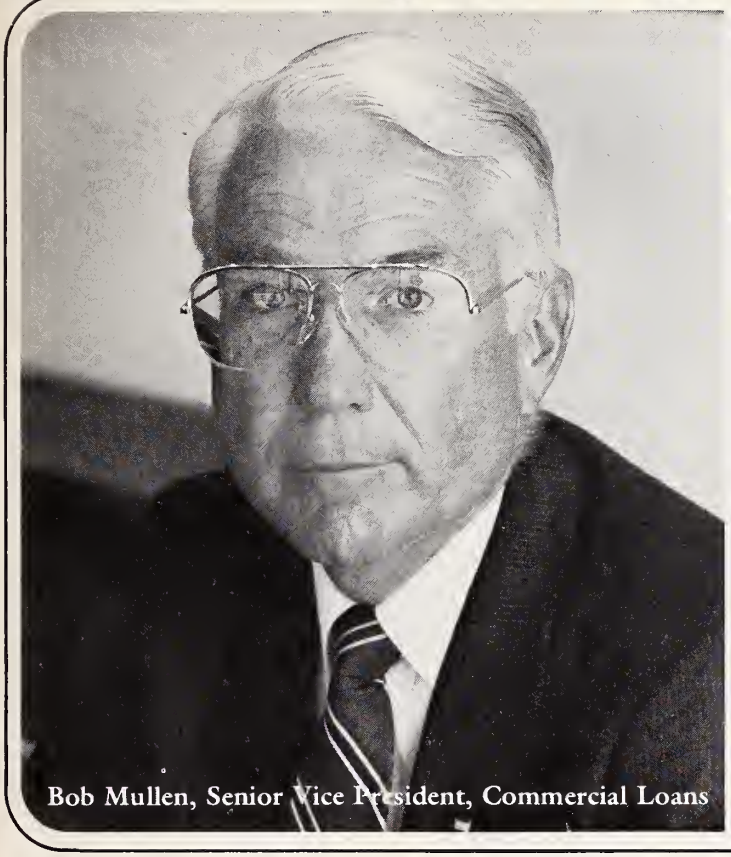


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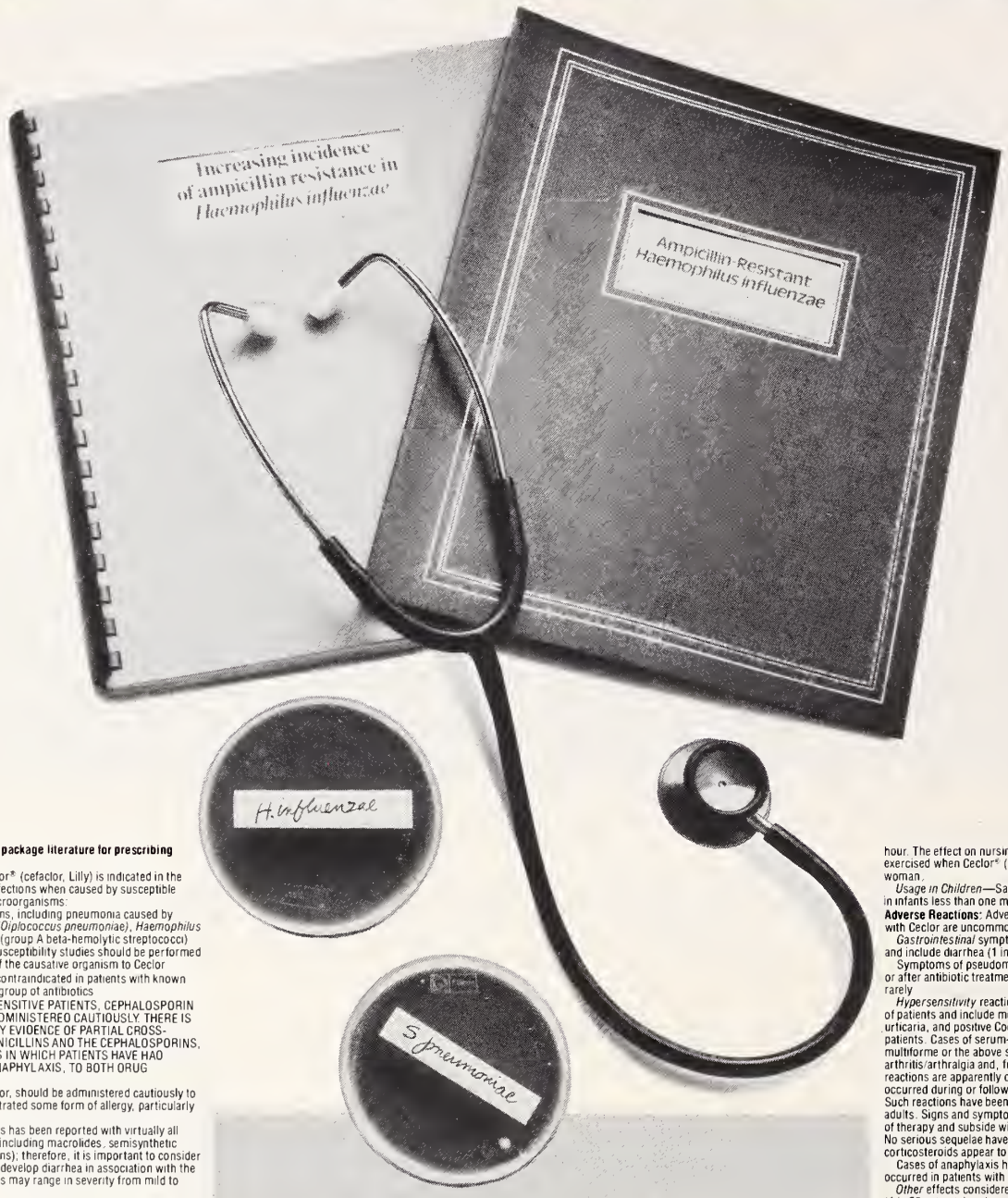
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An added complication... in the treatment of bacterial bronchitis*



Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

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Brief Summary. Consult the package literature for prescribing information.

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out.

Precautions: *General Precautions*—If an allergic reaction to Ceclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers—Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor® (cefaclor, Lilly) is administered to a nursing woman.

Usage in Children—Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below:

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations of SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

[0617B2R]

* Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

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KaMPAC MEMBERS 1983

The Kansas Medical Politial Action Committee was formed by the Kansas Medical Society in 1962 in order (1) to promote and strive for the improvement of government by encouraging and stimulating physicians and others to take a more active and effective part in governmental affairs; (2) to encourage physicians and others to understand and evaluate the nature and actions of government, the significance of political issues, the records of officeholders, and the pertinent qualifications of candidates for elective office; and (3) to assist physicians and others to organize themselves for effective political action to promote civic responsibility and integrity in government.

The Kansas Medical Society has a membership of 2,884 and the Kansas Medical Auxiliary has a membership of 1,274. The following physicians and spouses have elected to become members of KaMPAC and to support "organized medicine" in its efforts to elect "friends of medicine." This listing will be expanded each month to include new KaMPAC members.

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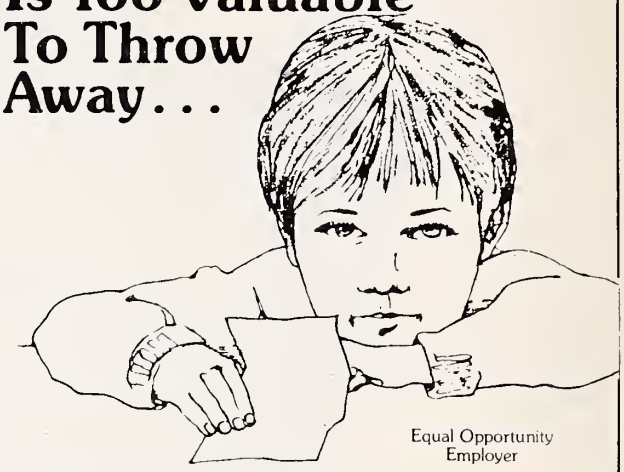
Vox Dox Editor:

In response to your request for input regarding Homebound/Hospital (H/H) instruction for pregnant females (*Journal,* November 1982), it is my feeling that these girls have demonstrated their health by getting pregnant — a state not usually gotten into by the severely ill. They should continue to demonstrate their healthiness by staying in school until they go into labor unless otherwise unable to do so because of complications of pregnancy such as eclampsia, twins requiring bedrest, etc., and — unless breastfeeding — should be back in school within two weeks. These are the usual behavior patterns for otherwise employed healthy females. Perhaps some consideration should be given to allowing them to breastfeed at school.

To do otherwise and not provide H/H for the father of the fetus (child) would appear to be sex discrimination and thus be unconstitutional.

David A. Leitch, M.D.
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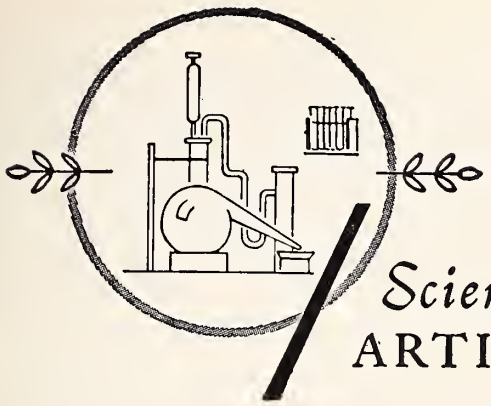
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Myasthenia Gravis

Its Pathophysiology, Diagnosis, and Treatment

JAMES T. LIN, M.D. *and* PHILIP A. SINGER, M.D., *Kansas City, Kansas*

DEVELOPMENT of effective immunotherapy for treatment of myasthenia gravis is one of the most exciting advances in neuroscience during the past decade. This fact, along with the development of animal model myasthenia gravis, the experimental autoimmune myasthenia gravis (EAMG) and subsequently the radioimmunoassay technique of antiacetylcholine receptor antibody,[1, 2] suggests strongly that myasthenia gravis indeed is an autoimmune disease. Myasthenia gravis has thus become an ideal model of human autoimmune disease in which specific antigen and antibody can be measured quantitatively. Antiacetylcholine receptor antibody (anti ach R) assay can be an invaluable tool for diagnosis and management of myasthenia gravis.

There is now strong evidence that myasthenia gravis is an autoimmune disease. Development of effective immunotherapy and various experimental techniques comprise exciting advances in neuroscientific research which are expanding our knowledge of this disease and its management.

Pathophysiology

In normal neuromuscular junction the presynaptic axon contains vesicles of acetylcholine (ach), the neurotransmitter *(Figure 1)*. Ach is synthesized by the enzyme choline acetyl transferase from choline and acetyl-CoA. Each vesicle contains about 10,000 acetylcholine molecules. In the resting stage, ach can be released through exocytosis into the synapse in vesicular form, so-called quantal release,[3] or in a nonquantal fashion. There are 200-1000 such vesicles close to the axonal membrane which are readily available for release. There is also a depot of acetylcholine vesicles adjacent to this immediately available pool. The matrix of the synapse contains acetylcholinesterase which inactivates and hydrolizes acetylcholine. The acetylcholine not destroyed by the enzyme is able to interact with acetylcholine receptor located on the postsynaptic membrane. There are tens of millions of such receptors per synapse. The interaction of acetylcholine and its receptor results in depolarization of the postsynaptic membrane by approximately one millivolt. With microelectrode recording, such depolarization by each quantum of acetylcholine vesicle can be measured and is called a miniature end plate potential (MEPP).[3] The frequency of MEPP in a normal human intercostal muscle is 0.2/second.[4] With volitional activation, or electrical activation of the axon, the arrival of the action potential along the axonal membrane will result in ejection of 50-200 acetylcholine quanta from the presynaptic membrane. This facilitated ejection of the ach vesicles requires Ca^{++} to bind to the presynaptic membrane and probably is mediated through cyclic adenosine 3′:5′ monophosphate (AMP). The summation of MEPP

From the Department of Neurology and the Ralph L. Smith Research Center, The University of Kansas Medical Center, Kansas City, Kansas; and the Veterans Administration Medical Center, Kansas City, Missouri.

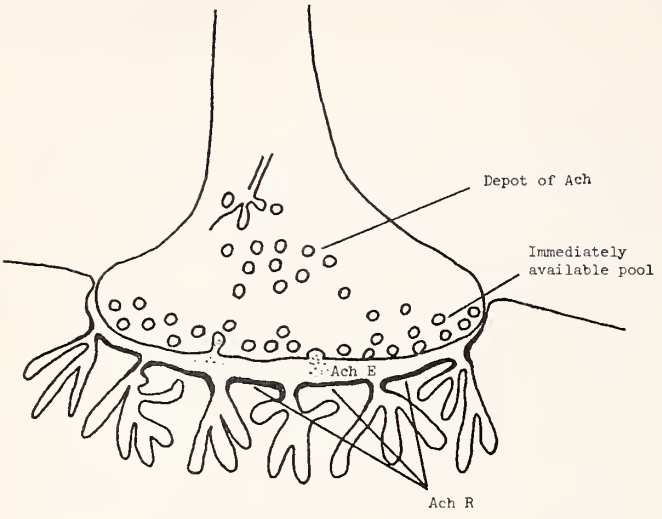


Figure 1. Simplified schema of the neuromuscular synapse. The receptors are shown as dark bands on postsynaptic membrane.

generated by each nerve impulse is called an end plate potential (EPP) which in general exceeds the threshold of postsynaptic membrane and thus produces a propagating action potential along the muscle membrane. The amplitude of MEPP and EPP are therefore proportional to: (1) the number of acetylcholine quanta available for release; (2) the probability of release from the presynaptic membrane — for example, the binding of the Ca^{++}; and (3) the number of acetylcholine receptors located on the postsynaptic membrane.[5] Under normal physiological conditions the safety factor of this neuromuscular transmission is about five times the required amplitude for the activation of the postsynaptic membrane. Repetitive volitional or electrical activation of the motor neuron will result in reduction of EPP after a brief initial facilitation due to the depletion of readily available acetylcholine store.[3] This will not, however, result in failure of transmission because of the huge safety margin. Any disease process that affects the presynaptic neurons, synaptic structure, or postsynaptic membrane may result in transmission failure if the safety factors enumerated above are impaired.

Presynaptic mechanisms that may result in the failure of neuromuscular transmission include myasthenic syndrome sometimes associated with bronchogenic carcinoma, or Eaton-Lambert syndrome.[6] This is probably due to the defective release of the acetylcholine vesicles. Studies with microelectrode technique has shown the amplitude of MEPP is normal but its frequency is decreased. Although the exact pathogenesis is unknown, there is some evidence to suggest that this may be the result of an autoimmune disorder.[7] Other presynaptic mechanisms include botulinum toxin, drugs, venom of the black widow, tick and scorpion, certain types of congenital myasthenia gravis, motor neuron disease, and peripheral neuropathies. Congenital myasthenia gravis with absent 16 S acetylcholinesterase,[8] organophosphate insecticides, anticholinesterase and such, will cause a defect in neuromuscular transmission due to postsynaptic mechanisms. Post synaptic failure may also be caused by myopathy, congenital myasthenia gravis, curare, tetracycline, d-penicillamine or other drugs, and most commonly, autoimmune myasthenia gravis.[5]

TABLE I
CLINICAL TYPES FOR MYASTHENIA GRAVIS

Neonatal myasthenia gravis — 12% of babies born to mothers with autoimmune myasthenia gravis
Congenital myasthenia gravis — heterogenous group
Autoimmune myasthenia gravis
Myasthenia gravis associated with polymyositis
Myasthenia gravis associated with Eaton-Lambert syndrome

In clinical practice one may therefore see different types of myasthenia gravis *(Table I)*. Neonatal myasthenia gravis occurs in newborn infants from birth to several weeks of life, and usually is self limited. About 12 per cent of babies born to mothers with autoimmune myasthenia gravis and with high antibody titer will contract this disorder.[9] Congenital myasthenia gravis is a heterogenous group of disorders.[10] In general, antiacetylcholine receptor antibody is not detected, and therefore treatment with steroids or other immune suppression is not indicated. There are also acquired myasthenia gravis, presumably on the basis of autoimmune process associated with polymyositis or Eaton-Lambert syndrome.[11] However, autoimmune myasthenia gravis is by far the most common.

Immunopathology

In 1973, Patrick and Lindstrom induced experimental autoimmune myasthenia gravis (EAMG) by immunizing rabbits with electric eel ach receptors.[1, 12] EAMG includes acute and chronic phases. The acute phase occurs 8-11 days after immunization and is probably due to T lymphocyte and macrophage activation with destruction of the end plate.[13] A chronic phase occurs 28-30 days later, with muscular weakness and clinical features similar to human myasthenia gravis. Subsequent study of EAMG has revealed the following characteristics:

- It can be induced in different animal species, although there is some species specificity;
- EAMG can be passively transferred;[14]
- It involves both T and B cell activation;[15]
- Antiacetylcholine receptor antibody can be measured; and finally,
- EAMG has both clinical and electrophysiological similarities to human disease.

Although humoral blocking factors for neuromuscular transmission had been postulated for quite some time, it was not until the development of the assay technique for antiacetylcholine receptor antibody that the role of antibody was clearly delineated. Anti ach R can be detected in 90 per cent of patients with myasthenia gravis.[16] It has been shown by immunochemical and immunopathological methods to be deposited on the postsynaptic membrane in myasthenia gravis.[17] Although the correlation between clinical disease and anti ach R antibody is poor, within any individual patient the reduction of anti ach R is often associated with clinical improvement.[18] It has also been shown that anti ach R antibody is of heterogenous origin and may represent a polyclonal activation.[19] Association of myasthenia gravis and other autoantibodies is well known.

Several mechanisms have been postulated to explain the presence of anti ach R and the clinical, pathological changes in neuromuscular junctions. The antibody may block the acetylcholine binding to the receptor and consequently induce the clinical weakness. This type of antibody may therefore be correlated with clinical severity of disease. Drachman *et al.*[20] have shown that the aggregation of acetylcholine receptors by antibody cross linkage could result in internalization and modulation of the postsynaptic membrane. Fixation of antibody and complement to the postsynaptic membrane can result in the destruction of the membrane and release of immune complexes.

The exact mechanism in which anti ach R antibody is activated is unknown in human myasthenia. The thymus has long been suspected of being the culprit. Seventy per cent of patients with autoimmune myasthenia gravis have evidence of thymus abnormality and 10 per cent of them have thymoma. Acetylcholine receptors and muscle-like cells have been noted in the thymus.[21] Small amounts of anti ach R antibody can be synthesized in thymus cell cultures from patients with myasthenia gravis.[22] More recently the immunoregulatory lymphocytes, particularly those of the T suppressor cell, have been implicated.[23] As many as 20 per cent of patients with

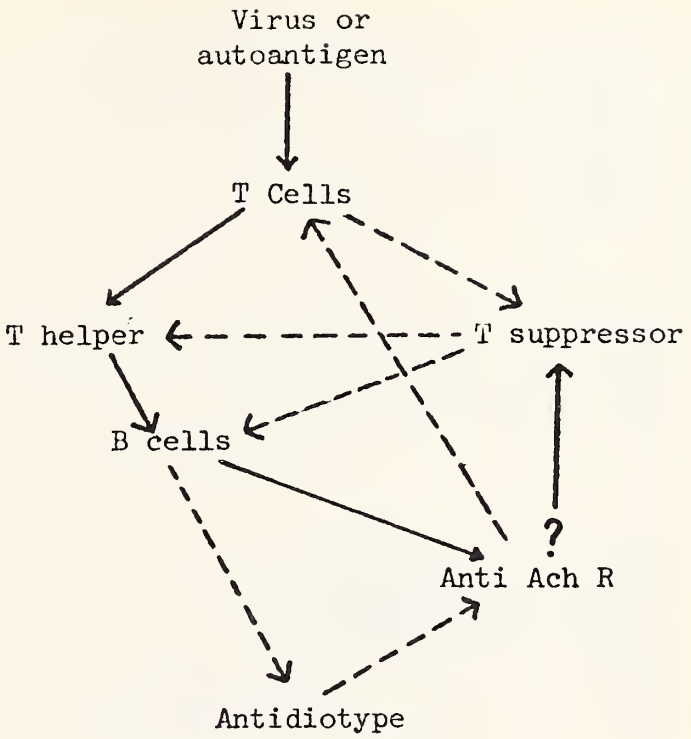


Figure 2. Possible immune mechanism for autoimmune myasthenia gravis. The solid lines suggest positive influence for the production of anti ach R. The broken lines suggest negative influence and may be related to the development of immune tolerance.

myasthenia gravis may go into spontaneous remission after several years. The immune tolerance may depend on the activation of T suppressor cell or probably the development of a second antibody in response to the anti ach R antibody — the so called antiidiotype antibody that can nullify the effect of anti ach R.[24] *Figure 2* illustrates this proposed network theory of immunoregulation in myasthenia gravis.

Diagnosis

The clinical diagnosis of myasthenia gravis is at times difficult. Bimodal sex and age distribution is frequently noted in females less than 35 years old and older male patients. Muscular weakness commonly occurs in ocular, bulbar, and proximal limb muscles. The ocular muscle weakness is often asymmetrical where the limb is proximal and symmetrical, although asymmetrical or isolated muscle weakness is not uncommon. Fatiguability is characteristic of this disease, but not always elicited from the history. If the disease is severe, the patient may not experience refreshment after rest. Muscular atrophy

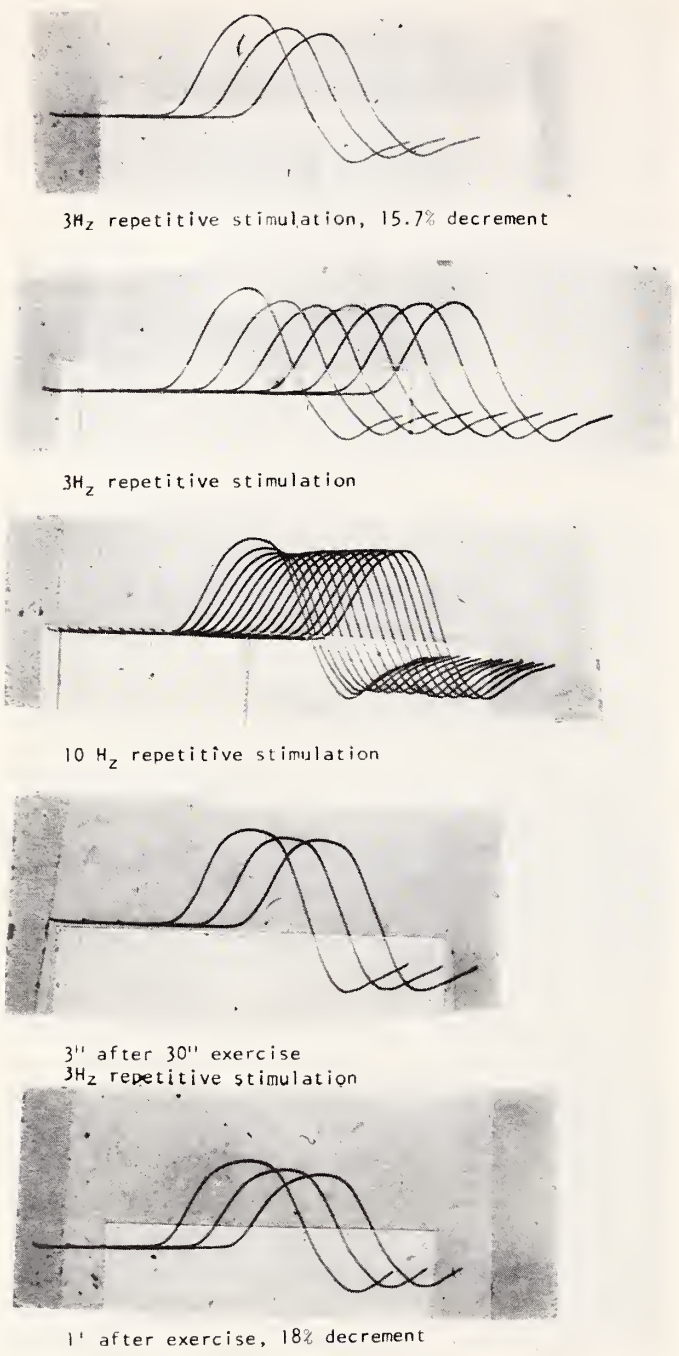


Figure 3. Repetitive nerve stimulation in a patient showing decremental response (top) which becomes improved shortly after exercise (facilitation). The decrement worsens again several minutes later (bottom).

is not a prominent feature, and sensory abnormality is absent. Deep tendon reflexes are normal.

Edrophonium (Tensilon) or Prostigmin can be used to demonstrate improvement in the weak muscle. The Tensilon used is usually 10 mg in a tuberculin syringe. Two mg is used as a test dose followed by slow push. Sometimes the Tensilon test can be equivocal in cases where the weakness cannot be measured objectively, and therefore a double blind test protocol has been suggested. However, in our practice the use of this technique does not yield any more information, nor can the use of Tensilon determine the optimal dosage of antiacetylcholinesterase in myasthenia.[25]

Repetitive nerve stimulation has become a standard test for myasthenia gravis. In the myasthenic patient, the decrease of EPP from repetitive nerve stimulation is often well below the threshold of postsynaptic membrane and therefore results in failure of synaptic transmission *(Figure 3)*. This kind of decrement can be improved slightly with exercise or by electrical tetanization (postactivation facilitation) for a few seconds due to enhanced ach release. Two or three minutes later, there is usually again decremental response greater than before exercise (postactivation exhaustion). The sensitivity of this test in a reliable laboratory is 60-95 per cent. In mild myasthenia gravis or ocular myasthenia, a single fiber EMG may demonstate the variability of neuromuscular transmission (jitter) and thus improve the diagnostic accuracy.[26]

Because of the specific association of anti ach R antibody and myasthenia gravis, the determination of antibody titer in suspected myasthenic patients has become extremely useful for both diagnosis and therapy. The demonstration of antibody will therefore implicate autoimmune etiology and justify the immune therapy.

The stapedius reflex based on testing the decay of the audiogenic reflex is painless and may become useful in the future.

Therapies

Clinical and laboratory evidence suggests autoimmune myasthenia gravis is not an homogenous disease process. The clinical course may vary considerably among individuals. For instance, ocular myasthenia gravis which has low antibody titer and variable response to anticholinesterase and poses no life threat is probably different from other forms of myasthenia gravis *(Table II)*. The natural course of autoimmune myasthenia gravis suggests that the first few years are most severe, and many patients may go into a remission spontaneously thereafter. Any therapeutic approach to myasthenia gravis therefore should include this consideration.

A modified Osserman classification of myasthenia gravis according to the severity and course of the disease has been useful for therapeutic planning. A rational classification based on age, HLA typing, antibody titer and presence of thymoma[27] *(Table II)*,

TABLE II
CLINICAL CLASSIFICATION

Modified Osserman
Ocular
Low EMG diagnostic yield and with low anti ach R titer
Generalized
Mild
Severe
Acute fulminating
Association of thymoma, high anti ach and anti striated muscle antibodies
Late severe
Chronic with muscle atrophy
Young age onset (40 years old) associated with HLA A1 & B8 — mostly female
Old age with HLA A3, B7 association
Association with thymoma

in conjunction with modified Osserman classification, may provide useful information in clinical practice.

Anticholinesterase

Anticholinesterase inhibits hydrolysis of acetylcholine so that interaction between acetylcholine and postsynaptic membrane receptor can be enhanced nonspecifically. Anticholinesterase therefore can be used for the treatment of myasthenia gravis regardless of the clinical stage. Although there have been reports that suggest anticholinesterase may induce deterioration of the synaptic structure, this requires a very high dose in animal studies. It appears that the destruction may be due solely to the antibody and complement attack of the postsynaptic membrane.

Anticholinesterase may be used alone in ocular myasthenia and in general myasthenia if the symptoms can be controlled adequately. Although the agent does not attack the basic pathogenic mechanism of myasthenia gravis, the majority of patients can be treated this way with no significant side effects, and with time some of them may go into remission due to the development of immune tolerance.

Pyridostigmine bromide (Mestinon) should be used first because of less gastrointestinal muscarinic side effect. The initial adult dosage is approximately 30-60 mg four times per day. Dosage may be adjusted and increased up to 400-500 mg daily. The drug is effective in 30-60 minutes and lasts up to six hours. For pediatric and feeding tube use, a syrup form of 12 mg/ml is available, as is a solution (5 mg/ml) for parenteral use. A slow release time span capsule is available for night use. If a patient develops excessive diarrhea or muscle cramps, atropine, 0.4 mg every four to six hours, or loperamide HCL (Imodium) may be used.

Over medication with anticholinesterase (cholinergic crisis) may exacerbate the clinical weakness. However, this complication is now infrequent. The short acting edrophonium (Tensilon) is of little use to determine whether the patient is under or over medicated. At times one may see under medication in some muscle groups and over medication in others within an individual. When the patient is critically ill and not responding to a reasonable dosage of antichloinesterase, s/he should be placed under close observation and with accessible respiratory support facility. If there is elevation of antiacetylcholine recepter antibody, plasmapheresis should be considered as a short term treatment and in preparation for thymectomy.

Thymectomy

There are several rationales for thymectomy in myasthenia gravis. The thymus may be the site of autoimmunity in myasthenia gravis. Ten per cent of patients with myasthenia gravis may have thymoma, which is usually associated with high anti ach R and antistriated muscle antibodies.[28] Because of the invasive nature of thymoma, thymectomy should be performed if there is any radiological evidence of enlarged mediastinum. Thymectomy has been shown to decrease the anti ach R antibody titer and induce clinical improvement in significant numbers of patients. Thymectomy is particularly recommended in young female patients with early myasthenia gravis. There have been no adverse effects in adults without thymus, and in our institution thymectomy is associated with almost no mortality. It appears, therefore, that thymectomy offers one time surgical risk with significant possibility for long term remission and improvement in myasthenia gravis.

Thymectomy is not without drawbacks. Although thymectomy is currently an acceptable treatment in myasthenia gravis, there has never been a control trial to evaluate its efficacy.[25] It may take a few years for thymectomy to become effective clinically and thus it is difficult to ascertain whether the improvement is due to thymectomy or merely spontaneous remission. If the network theory of autoimmunity in myasthenia is true, thymectomy may become a double edged sword, for thymectomy itself has been shown to decrease the suppressor cell activity in humans.[30] Nevertheless, thymectomy is recommended in autoimmune myasthenia in young patients whose symptoms cannot be adequately con-

trolled by medication alone, in patients with thymoma, and in patients considered good surgical risks. Transternal approach is preferred because of easy visualization of the mediastinum. Except for an unsightly scar, the surgical risk is not increased with this method.

Steroid Therapy

Use of corticosteroids has been shown to improve the clinical disease and produce eventual remission in 50-80 per cent of patients with myasthenia gravis.[29] Their use has been associated with a decline of antibody level. They can be used alone or in conjunction with anticholinesterase or other immunosuppressive agents in generalized myasthenia not adequately controlled by anticholinesterase alone. It is most efficacious in elderly male patients. In critically ill patients, corticosteroids may be used with plasmapheresis in preparation for thymectomy.

Although steroid treatment may become effective within a few days, some patients may not show improvement for several months. Earlier reports also suggest transitory worsening of myasthenia at the initiation of treatment. Because steroid therapy is a nonspecific immune suppression, and requires long term use in myasthenia gravis, the side effects are significant.[29] Furthermore, there has been only one controlled clinical trial of steroids consisting of only 13 patients.[31]

It is recommended that steroid therapy be given in patients with severe generalized myasthenia not responding to anticholinesterase alone. The patient should be hospitalized where an ICU facility is accessible. Starting a dose of 50-100 mg prednisone daily is recommended. After two to four weeks, or when some clinical improvement is evident, the prednisone should be tapered gradually to an alternate daily dose for maintenance. This treatment may last for at least one year, and during this time serum anti ach R antibody should be measured periodically.

Plasmapheresis

The demonstration of anti ach R in myasthenic patients and its possible pathogenic role in this disease has raised considerable interest in therapy with plasmapheresis. Plasmapheresis has been shown to remove the antibody effectively from patients and induce a clinical improvement as early as 24 hours following treatment.[32] This is most effective in acute fulminant myasthenia (stage III). Repeated plasmapheresis in conjunction with an immunosuppressive agent or thymectomy can improve long term clinical status.[33, 34] We recommend the use of a continuous flow cell separator to exchange 3-5 liters of plasma which is substituted with lactate Ringer's solution and human serum albumin. We do three daily consecutive plasmaphereses for at least three weeks. Antibody titer needs to be monitored before and after each plasma exchange. The decline of antibody titer is usually associated with clinical improvement.

Plasmapheresis has been performed in different centers with different protocols. Because of the cost, it is not readily accessible to every patient. Plasmapheresis has been considered by most authorities to be a temporary measure although long term maintenance therapy has been used in some places. The reported side effects of plasmapheresis are infrequent,[33] and include allergic reaction with skin rash, nausea, vomiting, fainting or hypotension, very nonspecific generalized aching and pain, and fever or symptoms suggestive of flu. Development of thrombosis or air emboli has been reported. Infection of shunt and thrombosis may occur. Hypocoagulation is infrequently a problem.

Nonspecific Immune Therapy

Azathioprine (Imuran), cyclophosphamide (Cytoxan), and methotrexate all have been reported to be efficacious in the management of myasthenia gravis.[25, 34] Because of their potential side effects, these agents should be reserved for those patients who do not respond to the aforementioned treatment modalities. They are not usually used alone; they are frequently used in conjunction with corticosteroids or plasmapheresis. There has been no control trial or any evidence to suggest that one agent is preferable to others. We use azathioprine, 2.0-2.5 mg/kg/day as the first drug because it involves fewer side effects than do the others. Periodic surveillance of hematological and liver function should be performed. Side effects of this therapy should be weighed against the benefits in each individual patient.

Specific Immune Therapy

Immune therapy discussed earlier is intended to decrease the antibody level by inhibiting both T and B cell functions. These measures may only suppress the disease activity, and evidence suggests that the memory of lymphocyte sensitization is still present. The reported improvement with this treatment may in fact be due to the development of immune tolerance. Fuchs[35] and Fuchs *et al.*[24] had shown that in rabbits, denatured acetylcholine receptor is nonmyasthenogenic and can suppress EAMG. The study of this reduced carboxymethylated acetylcho-

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Skin Cancer

Parameters for Treatment

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SKIN CANCER is the most common type of malignancy, and is more prevalent in males than females. It is estimated that there are more than 300,000 new cases each year.[1] It is thought that 40-50 per cent of all people who live longer than 65 years will have had at least one skin cancer. Skin cancers are often curable; therefore the annual death rate of about 4,000 is too high.[2, 3]

Ninety to ninety-five per cent of skin cancers occur in exposed areas of the body. Although clinical diagnosis by physical characteristics is accurate in 90 per cent of cases, biopsies are essential. Most skin cancers are either basal cell carcinoma, which almost never metastasizes, or squamous cell carcinoma which does so rarely.

Treatment

Skin cancers can be treated successfully by radiotherapy or surgical excision. Although many methods of treatment are available, no single treatment is best in all circumstances. Basal cell and squamous cell carcinomas are radioresponsive and radiocurable. Careless application of any method can produce poor cosmetic results or make a curable lesion incurable. There is equal probability of local success at a given site with either radiotherapy or surgery; thus the one that will yield the best cosmetic and functional results should be selected. Therefore, the choice between surgery and irradiation depends on the tumor site, degree of extension, previous treatment, and the probable cosmetic and functional results.

Radiotherapy

Radiotherapy is indicated as the preferred method of treatment when it can destroy the carcinomatous tissue selectively without mutilation or dysfunction with little or no visible sequelae. In other words, radiotherapy is indicated when the extension of the lesion into deep structures makes treatment by surgery too mutilative. This reflects the real superiority of irradiation over excision in that it preserves normal anatomy.

Skin cancer is the most common malignancy, affecting about 300,000 people each year. Skin cancer is rarely fatal, and can be successfully treated by radiation therapy or surgical excision. The authors believe radiation therapy to be the preferred method of treatment because of its flexibility and achievement of excellent cosmetic results when used properly.

In order to achieve good cosmetic and functional results, two important factors should be considered in radiotherapy: radiation quality and fractionation of the total dose.

Quality of Radiation: A carefully planned technique is required in order to deliver the maximum cancerocidal dose to the tumor while at the same time minimizing the dose to normal tissue. This is best achieved by delivering x-rays of homogeneous quality throughout the entire volume of tissue irradiated. An x-ray machine generating an electromagnetic energy at 100 KV, with added filtration of 1-3 mm of aluminum, is sufficient to treat most superficial skin malignancies. For moderately deeply infiltrating lesions, an energy of 140-400 KV with added filters of several millimeters of copper are used. For extensive lesions involving bone or cartilage, megavoltage radiation such as cobalt, radium, or cesium should be used. Also, electron beams of varying energies can be used effectively in treating some skin malignancies.

Fractionation: Proper fractionation of the total dosage is an important part of effective radiotherapy. The longer overall time and the greater number of fractions possible, the less the normal tissue reaction and the better the cosmetic results. More ad-

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vanced lesions require larger portals. Traenkel and Muloy[4] reported that a total dose of 4000-5000 rads delivered as single weekly doses of 1000 rads produced four times as many episodes of skin necrosis as 300-400 rads three times each week. Daily treatments even further decrease the radiation sequelae.[5]

Skin cancer is rarely fatal; hence, it may be taken lightly and treated hurriedly to minimize patient inconvenience. Large individual doses or single massive dose techniques are popular. The cosmetic results of these techniques are poor, although local control is good. The results obtained by this method may be acceptable in a few sites, but on the face, use of protracted multiple fractions to deliver the total dose is mandatory.

Dosage: About 4000 rads in 10-12 fractions with a portal size of 2-3 cm, is an adequate dose in controlling most skin cancers. When the lesion is located on the eyelid, inner canthus or cartilage, a more protracted course of therapy, such as 4200-5000 rads in three to four weeks, should give good cosmetic results. For advanced and deeply infiltrating lesions, an accumulated dose of 6500-7500 rads, with supravoltage therapy in seven to eight weeks, is required. Care should be taken to protect the normal sensitive structures.

Prognosis and Results

Properly administered, radiotherapy treatment should result in a local control rate of 95-99 per cent, with good cosmetic and functional results. The cosmetic results are directly related to the size of the primary lesion and radiotherapy technique. Fitzpatrick *et al.*[6] reported a local control rate of 95 per cent for both basal and squamous cell carcinomas (total 477 cases) with good to excellent cosmetic results in 70 per cent of the cases. Hansen and Jensen[7] reported an 84 per cent ten-year cure rate in 1198 skin cancers, treated by 30-60 KV x-rays. In 1951, Mosekilde[8] reported on 603 skin cancers followed for five years with 90 per cent of them cured. Burt *et al.*[9] reported a local recurrence rate of 7.3 per cent at five years and 11.1 per cent at ten years. Good to excellent cosmetic results were seen in about 70 per cent of their patients. In Cherowosky's series,[10] only ten of 395 (2.5%) skin lesions (both basal and squamous cell carcinoma) recurred after being treated by radiotherapy.

Summary

Skin cancer is highly curable by a variety of means. Biopsy is essential prior to implementation of any therapy. Irradiation is the treatment of choice in situations where preservation of normal tissue, either for cosmetic or functional reasons, is important. Radiation therapy, because of its great flexibility, can be adapted to skin lesions of all sites and sizes. If shielding precautions and the quality and quantity of radiation are individualized, control rates are good, and cosmetic and functional results are excellent.

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Myasthenia Gravis

(Continued from page 58)

line receptor (RCM ach R) and the current advance in monoclonal antibody technique may have specific therapeutic value in myasthenia gravis.

It is probable that the induction of immune tolerance may become extremely important in future research of autoimmunity and specifically myasthenia gravis. For example, antiidiotypic antibody — *i.e.* antibody against determinant of human antiacetylcholine receptor antibody — has been successfully raised in animals.[36] Its role and possible therapeutic implications remain to be determined.

References may be obtained from the authors.

Gestational Trophoblastic Neoplasia

A Review of New Concepts

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GESTATIONAL TROPHOBLASTIC neoplasia (GTN) is a term that includes hydatidiform mole, invasive mole (or chorioadenoma destruens), and choriocarcinoma. Although material presented here will deal primarily with the hydatidiform mole, no discussion of the mole would be complete without mention of the other trophoblastic diseases and their relationship to the mole. These diseases have been known since antiquity. Hippocrates, in 400 BC, described hydatidiform mole as "dropsy of the uterus." In AD 600, Aetius of Armida described a uterus "filled with bladder-like objects." These diseases are the outcome either of degenerative or neoplastic transformation of gestational trophoblastic tissue. They are related in that hydatidiform mole always precedes invasive mole, and precedes gestational choriocarcinoma in one-half of the cases. The diseases differ in their biologic behavior or malignant potential. In 80-90 per cent of patients, hydatidiform mole spontaneously regresses after uterine evacuation. Chorioadenoma destruens characteristically invades the myometrium but has only limited propensity to metastasize. Choriocarcinoma grows quickly and metastasizes early in its course.

GTN is unique because of:

- its continuous production and secretion of human chorionic gonadotropin (HCG) — a very accurate marker of tumor size and thus tumor response to treatment. It is also useful in following the status of patients after completion of therapy to screen for recurrent disease;
- its exquisite sensitivity to chemotherapy. Choriocarcinoma, with once a usually fatal outcome, was one of the first solid tumors found to be completely responsive to chemotherapy in a majority of cases. In the early 1960s, gestational choriocarcinoma apparently isolated in the uterus, was cured in only 40 per cent of patients treated by hysterectomy. Today, trophoblastic disease with metastases to the lung or pelvis is cured in 90-95 per cent of patients treated with chemotherapy; and
- the presence of paternal transplantation antigens foreign to the maternal host.

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A review of new concepts in gestational trophoblastic neoplasia is presented, with special emphasis on diagnosis, followup of patients, classification of patients, and new chemotherapeutic agents effective for this disease.

The generic term gestational trophoblastic neoplasia is preferable to hydatidiform mole, invasive mole, or choriocarcinoma in the clinical setting since management of these neoplasms is usually determined by evaluation of beta-HCG titers without always precise knowledge of the histologic diagnosis.[1] Criteria more important than the lesion's histologic appearance in selecting a treatment program include presence or absence of metastases, and when present, their specific location; duration of the disease; tumor volume as measured by beta-HCG titers; prior chemotherapy failure; and prior term pregnancy. Because of the importance of metastases in determining prognosis and treatment, GTN is broadly divided into metastatic (MTN) and nonmetastatic (NMTN) trophoblastic neoplasia.

Hydatidiform Mole

In most cases, hydatidiform moles develop within the uterine cavity. However, they may occur in any ectopic site of pregnancy. The uterus is filled with a delicate, friable mass of thin-walled, translucent, cystic, grape-like structures. On microscopic examination, characteristics of the mole include virtual absence of villus vasculature, hydropic swelling of chorionic villi, and variable degrees of hyperplasia and anaplasia of the trophoblast. The central substance of the villi is a loose, myxomatous, edematous stroma. The lining is chorionic epithelium, both

cytotrophoblast and syncytiotrophoblast. The degree of hyperplasia and anaplasia of the trophoblast tends to correlate with the risk of subsequent malignant sequelae; however, this is not an absolute factor for each individual patient. A mole with benign-appearing trophoblastic proliferation can undergo malignant change and, conversely, one with marked anaplasia and hyperplasia can regress spontaneously. Therefore, all patients with a histopathologic diagnosis of hydatidiform mole must be evaluated carefully and followed closely. It is unclear whether the morphologic changes seen are primarily degenerative, resulting from absent or inadequate fetal circulation, or neoplastic with low-grade malignant potential. The importance of hydatidiform mole relates to the risk of subsequent malignant sequelae.[1] Although only 10-20 per cent of women with moles require treatment, approximately 85-90 per cent of women needing chemotherapy have had prior molar gestation.

The incidence of hydatidiform mole varies greatly according to age, geographic location, and socioeconomic conditions. It occurs ten times more often in women over age 45 years than in those under age 40 years. In the U.S. about one in every 2000 pregnancies results in hydatidiform mole. In other developed nations, the incidence is similar. In less developed areas, including Mexico and Taiwan, the incidence increases to one in every 120 pregnancies. In the Philippines, one in 2000 pregnancies results in moles in the private patient population. In the Philippines during World War II, when there was widespread malnutrition, there was an overall incidence of one in 100 pregnancies. The incidence of gestational choriocarcinoma in Asians who move to the United States drops to a level between that of the two geographic locales. These data suggest that nutritional, environmental, and genetic factors all play a role in the etiology of this disease.

In early gestation, molar pregnancy is often clinically indistinguishable from a normal pregnancy. Early abortion frequently occurs, but not always. Hydatidiform mole should be considered in any woman with bleeding during the first two trimesters of pregnancy. Uterine bleeding is the single outstanding sign and occurs in more than 90 per cent of molar pregnancies. It can be intermittent or continuous, ranging from spotting to profuse hemorrhage. Because bleeding is a frequent and often nonspecific finding in early pregnancy, additional signs and symptoms are usually needed to make the diagnosis. Disproportionate uterine enlargement for expected gestational age is frequently seen. Approximately 45 per cent of such patients have a uterine size in excess of that expected; 16 per cent have uteri of normal size; and 38 per cent have uteri smaller than expected. Hypertension, proteinuria and other signs of pre-eclampsia, rarely seen in the first two trimesters of pregnancy, occur in 25 per cent of patients with molar pregnancies. Hyperthyroidism is seen in about 5 per cent. In 40-50 per cent, the diagnosis is promptly established by passage of characteristic clear, grapelike, hydatid vesicles. Disorders occasionally noted include intermittent lower abdominal pain, and hyperemesis sometimes requiring hospitalization. Absence of fetal parts on palpation and absence of fetal heart tones at a gestational age when they would be expected should lead to consideration of an abnormal pregnancy. Massive ovarian theca-lutein cysts may occur because of ovarian hyperstimulation by HCG. They are present in about 30 per cent of patients with hydatidiform moles.

Diagnostic studies can include abdominal radiographs, amniography, and sonography. Abdominal films are of help if the pregnancy is of 20 weeks or more duration. If a fetal skeleton is seen on x-ray, the diagnosis of hydatidiform mole is doubtful since coexisting fetus and molar gestations rarely occur. It is supported if a skeleton is not seen. The plain x-ray film is least sensitive of the three diagnostic studies mentioned so far. Amniography and sonography are accurate in more than 90 per cent of patients. In molar pregnancy, amniotic fluid is usually not present. Therefore, a dry amniotic tap during amniography suggests a hydatidiform mole. The characteristic honeycomb or moth-eaten pattern seen on x-ray after injection of radiopaque dye is diagnostic. Sonography is preferred in most centers because it has equal diagnostic accuracy, results in less discomfort, and has no risk of radiation exposure to a normal pregnancy. Sonography can also identify theca-lutein cysts. Beta-HCG determinations are mandatory in evaluating and following patients with GTN. They are not reliable in diagnosing hydatidiform mole until after the 14th week of pregnancy, when beta-HCG levels become low in normal gestation and remain high in molar pregnancy.

Once the diagnosis is confirmed, initial management should be uterine evacuation as quickly as possible since malignant sequelae are markedly increased in untreated patients. Suction dilatation and curettage (D&C) is the easiest and least morbid way to accomplish this, even in the presence of uterine enlargement. Concomitant use of oxytocin and ergotrate with curettage will reduce potential blood loss; however, because of decreased uterine sensitivity to oxytocin during the first two trimesters of pregnancy

and the frequent brisk hemorrhage associated with suction D&C, blood should be available for transfusion. Oxytocin administered prior to uterine evacuation apparently increases the risk of transport of molar tissue to the lungs, with resultant respiratory distress. Therefore, it should be given only after the uterus is evacuated. If further childbearing is not desired, management consists of total abdominal hysterectomy. Hysterectomy markedly reduces the risk of malignant sequelae and recurrences, although it does not eliminate them entirely. Thus, the same appropriate followup, with beta-HCG testing, must be carried out as for women treated initially with suction curettage. The use of hysterotomy in initial management should be discouraged. Recent studies suggest a slightly higher incidence of postmolar malignancy in patients whose primary evacuation is hysterotomy rather than hysterectomy or D&C. Removal of the ovaries is rarely necessary even with massive theca-lutein cysts since these will regress spontaneously following the decline of elevated HCG levels.

Some people have advocated the use of one course of prophylactic chemotherapy in managing patients at the time of uterine evacuation. This approach has not gained wide support since 80-90 per cent of these women are cured with initial uterine evacuation and therefore would not require chemotherapy. In addition, the prognosis for cure of patients with malignant sequelae who have been under close observation approaches 100 per cent making routine prophylactic treatment seem unwarranted.

Prognosis

Beta-HCG titers should be determined weekly following uterine evacuation of a hydatidiform mole. Eighty per cent of patients will have undetectable levels of beta-HCG eight weeks post-uterine evacuation. About one-half of the remaining patients (*i.e.* 10-15% of all patients) will have malignant sequelae (8-10% chorioadenoma destruens and 2-5% choriocarcinoma), and chemotherapy is administered to these patients. Likewise, patients in whom titers of beta-HCG rise or reach a plateau for two consecutive weeks following curettage should receive chemotherapy. To check for evidence of metastases, chest x-rays should be taken and pelvic examination should be done at the time of diagnosis and then monthly until beta-HCG titers are undetectable. Any patient whose examination reveals metastases should be treated immediately. Factors that increase the risk of malignant sequelae include hyperplastic and anaplastic trophoblastic tissue, age greater than 40 years, presence of theca-lutein cysts, and uterine size larger than expected for the length of gestation.

All women with GTN should undergo a complete evaluation prior to beginning chemotherapy. Lungs, vagina, and pelvis are the most common sites of metastases. Brain and liver metastases, which when present make the patient "high risk," occur in 10-12 per cent of patients. Therefore, a complete evaluation should include x-ray films of the chest, liver scan, liver function tests, brain scan, and neurologic and pelvic examinations.

Nonmetastatic tumor

If the workup for metastases is negative, chemotherapy should begin with either actinomycin D or methotrexate, which are equally effective drugs. Both drugs can cause nausea, vomiting, diarrhea, alopecia, skin rash, stomatitis, ulcerations in the gastrointestinal tract, and bone-marrow depression. Because actinomycin D does not cause the hepatic and neurologic damage sometimes seen with methotrexate, it has often been used as the drug of first choice. However, it has recently been shown that methotrexate, when used with citrovorum factor rescue, is the least toxic single agent chemotherapeutic regimen for management of GTN. Whichever drug is used, it is given intravenously daily for five days. This course should be repeated every other week until three consecutive weekly titers are undetectable. The last course is given when negative titers are achieved. If there is no consistent decline, and titers plateau or rise, the single drug should be changed (methotrexate *vs* actinomycin D) or combination chemotherapy should be initiated.

Low-Risk Metastatic Tumor

If metastases are found, treatment is determined by the sites of metastases, the duration of disease as measured from the termination of the prior pregnancy or mole evacuation, the tumor mass as determined by the beta-HCG titer, prior term pregnancy, and prior chemotherapy failure. Patients with metastases anywhere except in the brain or liver, in whom the duration of disease is less than four months and whose serum beta-HCG titer is less than 40,000 mIU/ml, with no prior chemotherapy or term pregnancy are considered low risk to fail single drug therapy and are treated with actinomycin D or methotrexate as described above.

High-Risk Metastatic Tumor

All other patients with MTN are high risk to fail single-drug chemotherapy. Treatment must begin with vigorous triple-agent chemotherapy including

methotrexate, actinomycin D, and chlorambucil. While the first two are administered intravenously, the latter is administered orally. All three drugs are administered for five consecutive days and are repeated every other week until three consecutive weekly beta-HCG titers are undetectable. The last course is given when negative titers are achieved. When brain or liver metastases are present, radiation therapy can help increase survival. Three thousand rads should be delivered to the whole brain and 2000 rads to the liver, at a rate of 1000 rads/week. When treated with this protocol, survival in high risk patients approaches 75 per cent. However, if high risk patients are treated initially with a single drug, the survival rate is markedly lower even if the appropriate therapy is later instituted. If triple agent chemotherapy fails, other multiple drug regimens (Bagshawe protocol, combination of cis-platinum, Velban, and bleomycin) can be instituted with the possibility of cure.

In patients whose beta-HCG titers become undetectable after chemotherapy, 90 per cent will remain in remission. The remaining 10 per cent will have a secondary rise in titer. Of patients who relapse, the majority will have a detectable beta-HCG titer within two months of the initial negative titer; 1-2 per cent will have a delay of up to one year. Therefore, it is imperative that all patients with GTN be followed on a monthly basis for not less than one year following the initial undetectable beta-HCG titer. Subsequent fertility in patients with GTN has been shown to be normal.

Chorioadenoma Destruens

Invasive mole is always preceded by hydatidiform mole and results from approximately 8-10 per cent of such gestations. Microscopically, it has the same histopathologic features as those seen in hydatidiform mole, but is distinguished by its invasion into the uterine wall. Uterine bleeding and abdominal pain are characteristic. Invasive mole is always associated with an elevated beta-HCG titer and ovarian theca-lutein cysts as in other GTNs. Extensive myometrial invasion can lead to spontaneous uterine perforation. In addition, perforation can occur as a result of instrumentation at the time of D&C. When perforation occurs, hysterectomy is indicated. Invasive mole can metastasize, especially to the vagina and lungs. Treatment, as with all GTN, is identical to that previously described, and is highly successful.

Gestational Choriocarcinoma

Gestational choriocarcinoma is an aggressive, epithelial tumor of trophoblastic tissue. It may accompany or follow any type of pregnancy. The majority of cases occur in the uterus; however, ectopic pregnancies provide additional sites of proliferation.

Its incidence in the U.S. is one in 30,000-40,000 pregnancies. Choriocarcinoma is preceded by the following conditions: 50 per cent arise in hydatidiform moles; 25 per cent in previous abortions; 22 per cent in normal pregnancies; and the remainder in ectopic pregnancies and other sites. Choriocarcinoma results from approximately one in 40 hydatidiform moles, in contrast to one in about 150,000 normal pregnancies.

Choriocarcinoma is a purely epithelial cellular tumor that grows by the abnormal proliferation of both cytotrophoblast and syncytiotrophoblast. Histologically, there are no chorionic villi, only sheets of trophoblast. The tumor penetrates the underlying myometrium and quite often invades blood vessels and lymphatics. Widespread metastases are characteristic of this tumor. Favored locales of spread, in descending order of frequency, include the lungs (50%) and vagina (30-40%), followed by the brain, liver, and kidney. However, because of this tumor's propensity to disseminate through the vascular system, any organ or tissue may be affected.

Choriocarcinoma must be suspected and ruled out in any woman with unexplained uterine bleeding following childbirth. It can be further suspected by the presence of measurable beta-HCG in the nonpregnant woman. When the histologic diagnosis is made, chemotherapy must be instituted immediately. The evaluation, treatment, and followup of patients with choriocarcinoma are identical to that described above for GTN following evacuation of a hydatidiform mole.

Conclusion

GTN is one of the cancers that medical science has been able to combat most successfully. When diagnosed and treated properly, it is a highly curable malignancy. With the great potential for cure that chemotherapy offers these patients, it is imperative that this disease be recognized and treated appropriately.

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Interstitial Cystitis

A Review of the Literature

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"A RARE TYPE OF bladder ulcer in women" was first described in 1914 by Guy L. Hunner.[1] This ulcer subsequently came to be known as the elusive ulcer, or simply Hunner's ulcer. It is the hallmark of the disease *chronic interstitial cystitis,* or simply *interstitial cystitis*. Presented here is a review of current literature concerning this poorly understood disease.

Interstitial cystitis is a relatively uncommon disease affecting the bladder of both women and men, although it is much more common in women. It is characterized by nocturnal and diurnal frequency and urgency as well as suprapubic and infrapubic pain, usually relieved or diminished by voiding. By definition, urine cultures are sterile, and results of urine analysis are nonspecific. Urine cytology yields negative results, and in many cases, further laboratory tests and diagnostic procedures are not helpful.

Nevertheless, the diagnosis has been made with increasing frequency in recent years. This is attributable both to increased awareness of the disease and to improved ability to document and treat urinary tract infections. If a patient presents with persistent irritative bladder symptoms in spite of repeatedly sterile urine cultures, a thorough investigation for the causes of lower tract irritation is indicated. One possibility is interstitial cystitis.

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The cause of interstitial cystitis is unknown. The multitude of treatment regimens attest to the enigmatous etiology of the disease. (Bowers and Lattimer described 37 conservative and seven surgical methods in 1957, and the options have increased considerably since then.)

Interstitial cystitis is a relatively uncommon and histologically benign bladder condition that affects women more often than men. It is thought to be an autoimmune disorder with other contributing factors. Emerging knowledge and therapies are contributing to earlier, more accurate diagnosis and improved treatment of this annoying affliction.

It has been suggested, although not documented, that there may be a psychiatric basis for the disease. Subjective observations by clinicians often compare these "nervous women" to patients suffering from ulcerative colitis. A significant proportion of both groups of patients have been noted to have personality disorders, often of a depressive and despondent nature. It seems more likely, however, that the psychiatric disturbances often noted in these patients are secondary to the chronic symptoms of the disease combined with frustration over multiple unsuccess-

ful attempts at therapy, rather than vice versa. Borque concurred when he wrote in 1951:

> We have all met, at one time or another, patients who suffer chronically from their bladder; and we mean the ones who are distressed, not only periodically but constantly, having to urinate often, at all moments of the day and of the night, and suffering pains every time they void. We all know how these miserable patients are unhappy, and how distressing bladder symptoms get finally to influence their general state of health, physically at first, and mentally after a while.[2]

Heredity does not appear to play a significant role in the development of interstitial cystitis. Of several hundred patients reviewed, one set of monozygotic twins has been noted. There was an interval of a few years between the onset of the disease in these twins, but both ultimately developed intractable disease requiring cystectomy. One incidence of a mother and daughter with the disease was also reported.

Throughout the years there has been some support for a neurogenic origin of interstitial cystitis. Differential sacral neurotomy was performed as early as 1954 in the treatment of interstitial cystitis. Since then posterior rhizotomy, sacral neurectomy, cystocystoplasty, cystolysis, and even a form of denervation via electrical stimulation have been used to interrupt nerve supply to the bladder. These forms of therapy will be discussed more completely later. The point here is that all of these procedures have been reported, at one time or another, to relieve the symptoms of interstitial cystitis, and often to result in increased bladder capacity. Characteristic histopathological changes in the sacral nerves of interstitial cystitis patients were described by Bohm and associates in 1956. It is felt by various authors that these findings suggest a neurogenic origin of interstitial cystitis, specifically a pathological activity in the nerve supply to the bladder.

On the other hand, the histopathological changes in sacral nerves described by Bohm have not been reproducible in subsequent studies. Also, long term results of denervation procedures have been, at best, inconsistent. In his 1980 paper espousing the cystolysis procedure, Freiha wrote:

> . . . these operations (*e.g.* sacral neurectomy) have never gained popularity and have been abandoned. They were extensive operative procedures and often resulted in sensory denervation of the perineum and/or interfered with the sphincter mechanism. Furthermore, they gave indifferent results.[3]

It should be noted that cystolysis itself is simply another form of bladder denervation. Because it is a relatively new procedure, long-term followup information is not available. It may turn out that this procedure also will give indifferent results.

The question seems to be whether the resolution of the symptoms of interstitial cystitis by various denervation procedures is due to elimination of impulses to the bladder travelling by way of pathologically altered nerve roots, or is the result of reduction of parasympathetic and sacral somatic outflow. The bulk of the currently available information supports the latter explanation. The origin of interstitial cystitis is probably not neurogenic.

Currently, the most popular theory of the etiology of interstitial cystitis is that it is an autoimmune disease. This has been the subject of several recent papers, and elements of an autoimmune connective tissue disease have indeed been documented in many cases.

Direct evidence of the nature of interstitial cystitis as an autoimmune disease was first provided in 1970 by K. J. Oravisto, M.D., of the University of Helsinki, Finland, when he published immunological laboratory observations. Considerable evidence has subsequently accumulated in this regard and it will be summarized here.

The occurrence of active or recurring chronic inflammation and destruction of tissue without continuing cause — such as trauma or infection — strongly suggest an autoimmune pathogenesis. These diseases, once initiated, tend to be self-perpetuating and are accompanied by frequent remissions and exacerbations. Interstitial cystitis is characterized by these features.

Ninety per cent of cases occur in females, which is characteristic of autoimmune diseases.

Histologic changes have been found by several researchers to be consistent with connective tissue disease. Lymphocytes, plasma cells, mast cells, and eosinophiles have all been noted in bladder biopsies. It is known that an accumulation of these immunologically competent cells is observed at some stage in all autoimmune diseases. Varying amounts of fibrosis and edema are also frequently seen.

An increase in the incidence of various allergic phenomena has been noted in interstitial cystitis patients compared with controls, *e.g.* drug allergy and hay fever.

Interstitial cystitis has been described in patients with systemic lupus erythematosis (SLE), rheumatoid arthritis, polyarteritis nodosa, scleroderma, and autoimmune thyroiditis. The occurrence of two separate connective tissue diseases in the same patient is known to be a relatively frequent phenomenon. Boye (1979) describes a case of intersti-

tial cystitis representing a local manifestation of SLE in the bladder.

Antinuclear antibodies have been demonstrated to be elevated more frequently in interstitial cystitis patients than in controls. Oravisto reports 53 per cent elevation in one series and 85 per cent in another as opposed to 8 per cent of controls.

Circulating bladder-specific antibodies and immune-complex deposition in the bladder wall of affected patients have been reported.

Finally, therapeutic methods employed in connective tissue diseases have sometimes been effective in interstitial cystitis. These include immunosuppression with azathioprine and the use of corticosteriods, either orally or infiltrated into the bladder wall.

In his 1980 review of the disease, Oravisto states:

> It is evident that interstitial cystitis may be regarded as an autoimmune disease. The very chronic course of the disease, the absence of infection, the pathological findings, the common occurrence of antinuclear antibodies as well as the response to certain modalities of treatment speak in favor of the autoimmune character of interstitial cystitis.[4]

As might be expected, this belief is not universal. Messing (1978) states that:

> Both the recurrence of the disease in the intestinal tissue of intestinal cystoplasties and the loss of symptoms in patients who have their urine diverted without cystectomy make it unlikely that interstitial cystitis represents an antibladder tissue autoimmune disease.[5]

Messing's study also failed to reveal increased antinuclear antibody levels, circulating antibladder antibodies, or immunoglobulins within the bladder of patients with interstitial cystitis.

Concerning circulating antibladder antibodies, it should be noted that even if they are appropriately demonstrated after absorbing the subject's sera with nonbladder tissue, they may not necessarily be responsible for concurrent bladder inflammation. The antibodies may have developed secondary to the inflammation, as heart-reactive antibodies are known to do after chest trauma, myocardial infarction, or cardiac surgery.

There is also conflict regarding histological changes in bladder wall as characteristic of connective tissue diseases. This is particularly noted in regard to the presence of mast cells. Some authors have attached considerable significance to the presence of mast cells in the bladder wall biopsy, even suggesting that they are pathognomonic of interstitial cystitis. Messing reports, however, that mast cells may be found in the walls of many normal bladders and yet be conspicuously absent in the bladder of affected patients.

Finally, azathioprine and corticosteroid therapy have provided only occasional relief or palliation of symptoms and are currently rarely used to treat interstitial cystitis.

It is obvious that much work remains to be done before this disease is fully understood. In spite of the lack of universal acceptance, the bulk of current evidence suggests that interstitial cystitis belongs to the group of autoimmune diseases. Autoimmune diseases are thought to occur when antibodies are formed against components of the body. There are two broad categories of autoimmune disease: organ specific or disturbed antigen diseases (*e.g.* thyroiditis, atrophic gastritis) and non-organ specific or disturbed tolerance conditions (*e.g.* SLE). However, between these two extremes is a group of diseases characterized by organ specific damage and non-organ specific antibodies. Interstitial cystitis is a member of this group.

The diagnosis of interstitial cystitis is traditionally based upon symptom complex, laboratory tests, cystoscopy findings, and histopathological review of bladder wall biopsy. In addition, clinicians place varying amounts of significance on urethral and anterior vaginal wall tenderness during pelvic examination.

The symptoms of interstitial cystitis are quite variable in severity, frequently nonspecific, and highly subjective. The most commonly noted symptoms are frequency of urination, nocturia, suprapubic pain, dysuria, diffuse abdominal pain, urgency, vaginal pain, hesitancy, gross hematuria, back pain and rectal pain, in approximately that order. The symptoms are often noted to be relieved, at least partially, by voiding. It has also been noted that the symptom complex will develop rapidly (during a few months), then stabilize and remain approximately the same for many years. Mild remissions and exacerbations are not uncommon, but only 11 per cent of these patients become spontaneously free of discomfort.

The differential diagnosis of bladder diseases for a patient who presents with these signs and symptoms includes chronic infection, carcinoma of the bladder, tuberculosis of the bladder, shistosomiasis, or malakoplakia. Chronic infection is easily ruled out with repeatedly sterile urine cultures. Carcinoma of the bladder is characteristically a disease of elderly males. Some authors feel, however, that all of these patients should be biopsied to rule out carcinoma. Dunn (1977) stated that, "It is important to recognize that carcinoma of the bladder may present with

the same symptoms and similar cystoscopic appearances (as interstitial cystitis). It is therefore vital to biopsy the affected areas in every case to exclude multiple submucosal carcinoma.''[6] A more practical approach would be routine urine cytology to rule out carcinoma and biopsy for this indication only if a suspicious area is noted on cystoscopy.

Tuberculosis can be ruled out with urine cultures for acid-fast bacilli and the absence of any signs of tuberculosis in other locations. Shistosomiasis or bilharziasis which infects the bladder is caused by the organism *Shistosomiasis haematobia*. It can be ruled out by the absence of the characteristic ova in the urinary sediment, in tissue obtained from bladder wall biopsy, or less frequently in the stools. This infection is seen mostly in tropical and sub-tropical areas.

Malakoplakia is a rare lesion in the bladder mucosa characterized by numerous mottled yellow and gray plaques and nodules. It is most frequently seen in malnourished females in association with coliform infection, tuberculosis, or sarcoidosis. It may be ruled out by the absence of plaques or nodules on cystoscopy as well as the absence of the characteristic Michaelis-Guttman bodies (calcospherites) on histological examination of the bladder wall biopsy specimen.

Other than pertinent negative urine cultures just discussed, laboratory tests are of little value in the diagnosis of interstitial cystitis. Urine analysis may show mild hematuria or pyuria. In Messing's data, however, 90 per cent of patients had fewer than five red blood cells per high power field, and 88 per cent fewer than ten white blood cells per high power field. As previously noted, there are several series that report an increased incidence of elevated antinuclear antibody (ANA) in interstitial cystitis. The antibodies are seen more frequently in a severe than in a mild disease. The titer is generally low: 1:10 in one-half of the patients, 1:20-1:80 in one-fourth, and 1:60-1:1,280 in one-fourth in one series by Oravisto. Other immune function laboratory tests have not been reported to be abnormal. Although urine analysis is routinely performed and an ANA titer may be interesting, these and other laboratory tests should never preclude cystoscopy in the evaluation of a patient for interstitial cystitis.

Because of the frequently normal findings on physical examination, the protean symptoms and the nonspecific laboratory results, cystoscopy is necessary to make the diagnosis of interstitial cystitis. Radiologic contrast studies of the urinary tract and cystometric pressure studies usually are not necessary. The necessity for bladder wall biopsy is a moot point. There are certainly cases in which biopsy is not indicated and even some in which it may be contraindicated. The decision to biopsy is best made by the individual physician based on the clinical history and cystoscopic findings of each patient. It has been reported that interstitial cystitis patients often have very thin-walled bladders which are easily perforated at the time of transurethral biopsy.

In his original 1914 description of the disease, Hunner wrote, ''There is nothing absolutely characteristic in the cystoscopic picture. Perhaps the most characteristic thing is the insignificance of the lesion as compared with the long duration and intensity of the patient's suffering.''[1] There have, however, been multiple descriptions of the cystoscopic findings. Hunner described small areas of slight hyperemia or inflammation which bled easily when touched with the cystoscope. These superficial ''Hunner's ulcers'' were often surrounded by slight smooth white scars of former ulceration. The Hunner's ulcer is still considered by many to be the main criterion for diagnosis in these patients. It is usually located in the bladder dome and is noted most easily on slight overdistention. These patients have also been frequently noted to have a reduced bladder capacity. Bladder volume may range from 300 ml to as low as 50 ml, even under general anesthesia. Bladder capacity is smaller in the non-anesthetized patient. These two cystoscopic findings — reduced bladder capacity and one or more Hunner's ulcers — establish the diagnosis of the classic form of interstitial cystitis.

The majority of patients who present with the characteristic clinical history probably do not meet these criteria. In 1978, Messing wrote,

> . . . we seriously question whether or not a reduced bladder capacity needs to be present and we are certain that the majority of patients, and virtually all of the early cases, do not have a Hunner's ulcer. Indeed, we believe that the synonymity of Hunner's ulcer with interstitial cystitis has done more to prevent recognition of this disease than any other single factor.[5]

Messing goes on to describe the appearance of multiple, pinpoint, petechial hemorrhages or glomerulations throughout the vesical mucosa on the second distention of the bladder. The glomerulations may coalesce to become hemorrhagic spots which ooze drops of blood. Their location and abundance may vary, although they are rare on the trigone. This phenomena appears to be consistently noted in interstitial cystitis patients. It rarely occurs, however, in patients with normal bladders or irritative bladder symptoms caused by other conditions, such as be-

nign prostatic hypertrophy and radiation of the bladder. Messing believes these multiple glomerulations after the first overdistention to be the hallmark of interstitial cystitis.

This disease appears to have several gradations of severity. There is no doubt that the classic disease exists — with Hunner's ulcer, severe symptoms, a small capacity (possibly fibrotic) bladder, and poor response to therapy. Milder or early forms also exist without the ulcer or the contracted bladder, and with less severe symptoms. It is important to recognize these cases, as they are the ones more likely to respond to therapy. Multiple glomerulations after the first overdistention in a bladder that can be distended to a reasonably functional capacity, may be a valuable clue to the diagnosis of these early cases. It must be realized, however, that this is a subjective finding. Any bladder may get petechial hemorrhages if it is overdistended enough.

The histopathology of interstitial cystitis is not pathognomonic. Various reports have described a thin or absent epithelium and submucosal edema with dilated, thin-walled capillaries. Varying degrees of stromal fibrosis and fibrosis of the muscular layer have been noted. Chronic inflammatory changes are often seen in the tunica propria and muscularis. Plasma cells, lymphocytes, eosinophiles, and mast cells are reported in these inflammatory areas. There are several discrepancies in these descriptions. The only consistent characteristic is submucosal edema and vasodilation. The frequently mentioned pancystitis is an inconsistent finding, even in bladders with the classic cystoscopic disease. Considering the nonspecificity of these results, bladder wall biopsy is more likely to be useful in ruling out carcinoma in situ, tuberculosis, and shistosomiasis, than in making the diagnosis of interstitial cystitis.

Because there are no well-established objective criteria for diagnosing the disease, epidemiological statistics vary greatly. Onset of the disease is most frequently documented between the ages of 30 and 59 years. However, it occurs in persons of all ages. Lapides reported in 1975 that " . . . interstitial cystitis probably occurs as frequently in teenage females and in the third decade of life as it does in menopausal women."[7] The disease does not gradually progress in severity, but rather it tends to develop rapidly to its final grade, and then progress no further. The median duration of the disease before diagnosis is about four years, regardless of severity.

In 1975, Oravisto reported a prevalence of 18.1 cases/100,000 women of all ages. The joint prevalence of both sexes together was 10.6 cases/100,000. The annual incidence of new female cases was 1.2/100,000 women. Between 1975 and 1978, Freiha reported an incidence of one in 24 in his outpatient urology clinic. This is the highest incidence reported, probably due to diagnostic criteria, referral pattern, increased awareness, and earlier recognition of the disease. As mentioned earlier, the disease occurs ten times more frequently in women than men. In spite of the wide range in these statistics, it is safe to say that every urologist can expect to see at least one case every year. How should he treat it?

As Anthony Walsh so eloquently stated in the 1978 edition of *Campbell's Urology,* "An extraordinary gallimaufry of pills, potions, and procedures has been used in the attempt to treat this disease."[8] This, of course, reflects the unknown etiology of the disease and the frustration that has resulted from nearly all forms of treatment attempted. Generally, these forms of treatment may be classified as systemic, local, denervation, or bladder substitution.

Systemic therapy has been attempted with peptone, neoarsphenamine, emetine, oral ipecac, estrogens, potassium iodide, various vitamins, chloroquine, benzydamine, orgotein, oxyphenbutazone, antihistamines, anticholinergics such as Ditropan and ProBanthine, azathioprine, and corticosteroids. With only the latter two have any good results been well-documented during the last decade. The proponents of the auto-immune etiology have naturally tried the immunosuppressive and antiinflammatory drugs. Oravisto reported complete pain relief in 22 of 38 patients treated with azathioprine in one study. Steroids have been used with varying degrees of success by many workers. Badenoch reported sustained symptomatic improvement in 19 of 25 patients with oral steroids and recommended that all patients receive a trial with this therapy. More frequently, results have been entirely unpredictable, and the side effects from a course of treatment at the dose and length of time required have discouraged the therapeutic choice of oral corticosteroids.

Many forms of local treatment to the bladder have been tried. By far the most commonly and successfully used modality in the treatment of interstitial cystitis is simple distention of the bladder. This procedure nearly always provides at least partial relief of symptoms. The relief is often short-lived (one to three months), however, and only rarely does it continue for many months or a few years. Bladder distention may be used as the only form of treatment for mild disease. More commonly it is combined with other local therapy (infiltration or instillation). In an interesting study in 1977, Dunn reasoned that if

simple bladder distention produces short-term relief of symptoms, prolonged bladder distention might result in prolonged relief of symptoms. He distended the bladders of 25 patients to a pressure equal to their systolic blood pressure for two to three hours. Sixteen of these patients were symptom-free and six were symptomatically improved at an average of fourteen months. Prolonged bladder distention may have a place in the treatment of this disease.

Infiltration of the bladder wall, both with corticosteroids and with local analgesics, has been used. Oravisto has reported some success with intramural prednisolone, repeated at intervals of a few months, in patients with moderately severe disease. It is used in conjunction with bladder distention.

Transurethral resection and fulguration of bladder lesions is rarely advocated today. Hunner recommended resection of the ulcer in his original description of the disease. All current evidence, however, points to interstitial cystitis as a generalized panmural bladder disease. If this is true, resection and fulguration is not a theoretically sound treatment. Walsh makes the point that recent reports espousing this approach may have reflected symptomatic relief resulting from the prolonged bladder distention required to accomplish the procedure.

There have been about as many different chemicals instilled into the bladder as there have been systemic therapies attempted in the effort to treat this disease. Silver nitrate, Chlorpactin, and dimethylsulfoxide (DMSO) are the most frequently used today. Silver nitrate has been used in this way for more than a century. DeJuana (1977) advocates light fulguration of ulcers plus overdistention with silver nitrate solution. He claims good or excellent results (no retreatment for at least one year) in more than 50 per cent of patients. Simple distention may do as well.

Chlorpactin instillation under low pressure with moderate hydraulic distention is the treatment advocated by Messing and Stamey. Chlorpactin WCS-90, oxychlorosene sodium, is used in a 0.4 per cent solution. The instillations are extremely painful and must be performed under general anesthesia. The recommended treatment schedule consists of six instillations. The first two are followed by four weeks of observation for subsidence of symptoms. If symptoms persist, four further weekly instillations are recommended. These authors claim a 72 per cent success rate in achieving a symptom-free (or almost symptom-free) state for at least six months after the final instillation. Again, some of the results may be attributable to hydraulic distention. Messing has also reported one case of ureteral fibrosis secondary to Chlorpactin therapy and suggests that vesicoureteral reflux be ruled out before this therapy is initiated. Finally, some patients suffer severe irritative bladder symptoms for 24-48 hours after instillation. Although this can usually be controlled with oral narcotics, some authors have felt that the pain produced by Chlorpactin is disproportionate to the possible benefit received.

The most recent addition to the bevy of intravesical instillations is dimethylsulfoxide (DMSO). DMSO is by no means a new drug. It was first synthesized in 1867 and has been available as a by-product of the paper pulp industry for many years. It is now used chiefly as an industrial solvent but its medicinal properties have been known for many years. In 1965, however, testing of DMSO was curtailed by the Food and Drug Administration (FDA) following reports that it caused adverse ocular effects in laboratory animals. In 1978, a 50 per cent aqueous solution of DMSO was approved by the FDA for bladder instillation in interstitial cystitis patients. Among the wide range of its known pharmacological effects, the actions of membrane penetration, analgesia, anti-inflammation, bacteriostasis, vasodilatation, and dissolution of pathologic deposition of collagen make DMSO a theoretically effective agent in the treatment of interstitial cystitis. The recommended treatment regimen is 50 ml of 50 per cent solution instilled in the bladder for 15 minutes; the bladder is then emptied by voiding. Treatment is repeated every other week for four weeks, then as needed to control symptoms. Instillation may be done as an outpatient procedure or under general anesthesia for hydraulic bladder distention and cystoscopy. Side effects are usually limited to a garlic-like taste and odor on the breath and skin. The taste may last several hours and the odor for as long as 72 hours.

DMSO is reported to result in complete or near complete resolution of symptoms in 50-70 per cent of patients. The duration of relief is usually only one to three months. In some patients, however, it has had a dramatic and lasting effect. In light of the absence of serious side effects noted at this time and the ease of administration as an outpatient procedure, intravesical DMSO therapy should be recommended to those patients who have failed to respond to other conventional forms of therapy, and to those who present initially with severe irritative bladder symptoms.

It is not surprising that with the frequent ineffectiveness of conservative treatment for this disease, several surgical therapeutic approaches have been attempted. These can be generally classified under

the headings of denervation and bladder substitution. Denervation via sacral neurectomy and similar procedures has been briefly discussed under the section on neurogenic etiology. Although differential sacral neurectomy is generally in disrepute, it is still occasionally used and some well-documented reports of good results exist. Further trial of differential sacral neurectomy may be justified.

A more recent approach is supratrigonal denervation. This was originally accomplished in 1967 by cystocystoplasty. In this procedure the bladder is completely divided just above the trigone. All inferior and lateral communications are divided, including the inferior vesical vessels. Then the supratrigonal bladder is resutured to the base. In 1973, Worth described the less extensive procedure of cystolysis with the same goal in mind. In cystolysis, supratrigonal denervation is achieved by dissecting the bladder free of all surrounding tissues down to the trigone. Cystolysis is the operation of choice for supratrigonal denervation. As with any denervation procedure, it will not be effective if the bladder has undergone contraction and fibrosis. Freiha considers cystolysis only if the bladder can be distended to a volume of 400 cc or greater under general anesthesia. He reports on eleven cystolysis patients in which all have achieved partial or total relief of pain, and a decrease of frequency and nocturia. Freiha states that:

> The advantage of cystolysis over the other (denervation) procedures is that it offers the selective denervation of the supratrigonal portion of the bladder without interfering with the sphincter mechanism or with the bladder base sensations. This is evidenced by the fact that the urethral pressure profile remains unchanged postoperatively and that the patients do not lose the awareness of bladder distention and the desire to void.[3]

Also, the cystolysis procedure does not compromise subsequent cystoplasty should it become indicated. Cystolysis appears to be the denervation procedure of choice at this time. As mentioned earlier, however, long-term followup data are not available on the procedure.

A new form of treatment described by Fall (1980) is interesting and deserves brief mention. He used electrical stimulation to treat 14 patients with long-standing interstitial cystitis with severe symptoms. When bladder hyperactivity symptoms predominated he used intravaginal stimulation; when suprapubic pain predominated he used transcutaneous stimulation. Fall feels that the method is comparable to surgical denervation of the bladder since the effect of electrical stimulation is an inhibition of activity in the pelvic nerves supplying the bladder. Whatever the mechanism, frequency and pain were substantially reduced in all 14 patients.

The most aggressive way to treat interstitial cystitis is by bladder substitution. This includes the various types of enterocystoplasty as well as urinary diversion. Cecocystoplasty is currently the surgical treatment of choice for the small contracted bladder secondary to interstitial cystitis. Enterocystoplasty was first performed with ileum. Colon, and most recently cecum, were then determined to be easier and more effective for bladder augmentation. Properly performed cecocystoplasty may have very gratifying results. Various authors currently report 65-80 per cent symptomatic cures with the procedure. Lack of success with, and consequently poor acceptance of, the procedure may be partially attributable to misconceptions concerning its purpose. According to Walsh:

> In the treatment of interstitial cystitis, the object of enterocystoplasty is not enlargement but replacement of the diseased body of the bladder. Many authors have laid far too much emphasis on bladder capacity in this disease. Pain may be quite intolerable even though the bladder can be filled to 300, 400, or even 500 ml under anesthesia.[10]

Walsh goes on to emphasize that, except for the trigone, the entire bladder should be replaced. Any remaining diseased bladder wall may continue to cause pain, regardless of the expanded bladder capacity. Chan and Dounis, however, in two 1980 articles concerning cecocystoplasty, have found no correlation between the amount of bladder removed and the outcome of the operation. In all cases dissection should not be carried down behind the trigone, as this introduces a risk of nocturnal incontinence.

Because the intestinal graft is not as powerful as normal detrusor muscle, voiding can be a problem secondary to enterocystoplasty. Chan recommends a concurrent Y-V plasty with the procedure to assure bladder neck patency. Any prostatic adenoma must be enucleated in men and urethral dilatation may become necessary in women if they develop postoperative urinary retention.

Enterocystoplasty is recommended only in those patients with severe pain and contracted bladders whose conditions cannot be managed in a more conservative manner. Patients must be informed that their symptoms may recur and that they may even end up requiring lifelong intermittent catheterization. Nevertheless, it is a legitimate option which

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Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Antimicrobial Treatment of Systemic Fungal Infections

WAYNE BRABENDER, M.D. *and* GLENN R. HODGES, M.D., M.S., *Kansas City*

MANAGEMENT of systemic fungal infections is of concern to many physicians. Some fungal infections, such as acute pulmonary histoplasmosis, are often self limited and do not require treatment. However, normal hosts can develop systemic fungal infections requiring treatment, such as disseminated histoplasmosis or blastomycosis. Recently, opportunistic infections caused by less virulent organisms, such as *Candida* species, have been reported at a rapidly increasing rate. This increase is related to the use of invasive procedures, significant immunosuppression, and broad spectrum antibiotic therapy. The various chemotherapeutic modalities available to treat systemic fungal diseases are reviewed here. The entire spectrum of therapy is outlined in *Table I*.

Amphotericin-B

The polyene antifungal agents bind to cell-membrane sterols and cause changes in cell-membrane permeability leading to cell injury. Because of problems with stability, solubility and toxicity, only a few polyenes are clinically useful. Amphotericin-B (Fungizone), introduced in 1956, is the only polyene available for intravenous use. It is useful in treating aspergillosis, blastomycosis, candidiasis, coccidioidomycosis, cryptococcosis, histoplasmosis, paracoccidioidomycosis, and sporotrichosis.

Dosage Regimens. Dosage schedules are based on the limits of toxicity and somewhat on convenience. In most patients, an initial test dose of 1 mg is given due to the risk of severe hypotension or anaphylaxis. If the patient tolerates the test dose, then the dosage is increased by 5 mg every three days until a dosage of 0.5 mg/kg/day is reached. In critically ill patients, such as those with *Candida* septicemia, an accelerated program of administration can be used. After the 1 mg test dose, 0.25 mg/kg can be given the same day. If the patient tolerates that dose, 0.5 mg/kg/day can be administered starting the next day.

Certain selected patients can be treated with amphotericin-B as outpatients. Infusions of amphotericin-B are given on alternate days three times per week. These patients can often tolerate up to 1.0 mg/kg/infusion with no more toxicity than patients undergoing the more conventional daily infusions. The clinical response to amphotericin-B appears to be equal for both regimens.

There is little objective data on the appropriate length of amphotericin-B therapy for most systemic fungal infections. A cumulative dose of 2.0-2.5 grams of amphotericin-B during an 8-12 week period seems to significantly reduce the number of relapses when compared to smaller total dosage. In some instances, though, shorter courses may be efficacious. *Candida* species sepsis that is secondary to infected intravenous lines seems to respond well to a 300-500 mg cumulative dose if the infected lines are removed and there is no evidence of candida endophthalmitis at the conclusion of treatment. If endophthalmitis is detected, 2.0-2.5 grams of amphotericin-B are indicated.

Amphotericin-B is available in vials mixed with deoxycholate. This bile salt complex forms a suspension rather than a solution when hydrated, and

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TABLE I
DRUG THERAPY FOR SYSTEMIC FUNGAL INFECTIONS

Drug	*Infection*	*Usual Dosage*	*Metabolism*	*Major Side Effects*
Amphotericin-B[a]	Aspergillosis,[b] Blastomycosis, Candidiasis,[c] Coccidioidomycosis, Cryptococcosis,[d] Histoplasmosis, Paracoccidioidomycosis, Sporotrichosis	0.5 mg/kg/day IV; 30 to 40 mg/kg total dose; no change needed in chronic renal failure	Unknown; minimal liver and renal	Renal toxicity, anemia, hypokalemia, anaphylaxis (see text)
5-Fluorocytosine[e]	Candidiasis,[b, c] Torulopsosis,[b] Cryptococcosis,[d] Aspergillosis[b]	150 mg/kg/day in 4 divided doses po; lengthen interval if renal function decreased (see text)	Urinary excretion	Thrombocytopenia, leukopenia, anemia
Miconazole[a]	Coccidioidomycosis,[b] Candidiasis,[b] Paracoccidioidomycosis, Cryptococcosis[b]	400-1200 mg every 8 hrs IV; no adjustment needed for renal failure	Liver	Anaphylaxis, leukopenia, thrombocytopenia, hepatitis, hyponatremia
Ketoconazole[f]	Mucocutaneous Candidiasis,[b, c] Histoplasmosis,[b] Paracoccidioidomycosis, Coccidioidomycosis[b]	200-400 mg/day po; no adjustments needed for renal failure	Liver	Gynecomastia, fulminant hepatitis (?)

[a] For central nervous system infections, usually needs to be given parenterally and intrathecally.
[b] Efficacy in individual patients variable.
[c] See text for indications for treating candidiasis.
[d] 5-fluorocytosine used in combination with amphotericin-B.
[e] When used alone, organisms readily acquire resistance to 5-fluorocytosine during therapy.
[f] Should not be used for central nervous system infection because of poor cerebrospinal fluid penetration.

in-line filters should be avoided when amphotericin-B is administered. Only 5% dextrose — not saline — should be used to reconstitute the powder. Each dose should be given as a constant infusion during a period of three to four hours to avoid possible cardiotoxicity.

Because amphotericin-B levels in some body cavities are only one-half or less of levels in the serum, instillation of the drug directly into these areas sometimes supplements intravenous administration. Amphotericin-B can be given by the intrathecal route at lumbar, cisternal, or ventricular reservoir sites to treat meningeal infections with fungi such as *Cryptococcus neoformans* and *Coccidioides immitis*. From 0.1 to 0.5 mg of amphotericin-B should be diluted in 5 ml of sterile water or cerebrospinal fluid and infused slowly. Injections should be given no more frequently than 2-3 times per week. Intrathecal administration has been accompanied by headaches, pain in the back or legs, sensory loss, parasthesias, peripheral neuropathy, urinary retention, and impaired vision. Aseptic meningitis or an arachnoiditis can develop. Most of these side effects are reversible if administration of the drug is terminated. Intraarticular injections have been given for treatment of joint infections with fungi such as *Sporothrix schenkii*. Severe local reactions often occur with the recommended 5-50 mg doses and there is considerable difference of opinion as to whether intraarticular amphotericin-B should be used.

Toxicity. Amphotericin-B has numerous side effects. Almost all patients will experience chills, fever, nausea, and vomiting. Some patients will complain of diarrhea, malaise, and occasional muscle and joint pain. Severe hypotension and anaphylaxis occur occasionally. The less serious side effects will often abate with the continuation of the

drug even at higher doses. If these symptoms and signs are intolerable to the patient, premedication with aspirin or diphenhydramine will often help. Corticosteroids have been used, but if corticosteroids are given with the drug, amphotericin-B may bind to the corticosteroids with a subsequent decrease in the amount of drug available. There is no data to support the addition of heparin to the intravenous solution to decrease the incidence of phlebitis.

Almost all patients will develop normocytic, normochromic anemia during treatment with amphotericin-B due to decreased erythropoietin production. Iron therapy is usually not helpful and transfusions are rarely necessary. The hematocrit returns to normal within several weeks after cessation of therapy.

Renal toxicity occurs in all patients. A distal tubular acidosis with loss of bicarbonate, potassium, and water can cause significant hypokalemia in about 25 per cent of patients and requires potassium supplementation. Progressive elevations of serum creatinine and blood urea nitrogen will occur. Serum creatinine should be measured three times weekly and therapy temporarily suspended or the dosage decreased if the creatinine level rises above 3.0 mg/dl. Infusions can be given to patients on an every other day schedule to keep the creatinine below 3.0 mg/dl. After therapy is completed, serum creatinine and blood urea nitrogen will return toward pretreatment levels. However, even five years after treatment, as many as 75 per cent of patients will show some elevation of serum creatinine above pretreatment levels.

5-Fluorocytosine

5-fluorocytosine (Flucytosine) is a fluorinated pyrimidine initially developed as an antimetabolite for use in cancer chemotherapy and introduced in 1964 as an antifungal agent. Clinically, 5-fluorocytosine is active against yeasts such as *Candida* and *Torulopsis* species, *C. neoformans,* and *Aspergillus* species. However, it is rarely used as a single agent to treat infection caused by these organisms because resistance develops very quickly. The major use of 5-fluorocytosine is in conjunction with amphotericin-B to treat cryptococcal meningitis. Cerebospinal fluid levels reach 50-70 per cent of simultaneous serum levels. In combination with amphotericin-B, 5-fluorocytosine is often used to treat severe *Candida, Torulopsis,* and *Aspergillus* infections.

5-fluorocytosine is given orally due to very good absorption from the gastrointestinal tract. It is excreted virtually unchanged in the urine, which means serum levels are significantly influenced by changes in renal function. One recommended dosage schedule is 150 mg/kg/day divided by the serum creatinine and given in four doses per day. A second schedule is 150 mg/kg/day in four divided doses given every six hours and the interval is changed as the creatinine clearance (CrCl) changes. If the CrCl is 20-40 ml/min, the interval is changed to every 12 hours; if the CrCl is less than 20, the interval is changed to every 24 hours.

Common side effects are nausea, vomiting, diarrhea, and rash. About 5 per cent of patients will develop significant thrombocytopenia, leukopenia, or anemia. Many of these side effects are associated with serum levels greater than 100 μg/ml. These levels may be reached if the patient develops decreased renal function during therapy. All side effects are reversible if administration of the drug is stopped.

Imidazoles

Miconazole. The antifungal activity of imidazoles is by interference with the synthesis of sterols in fungal cell walls. Miconazole (Monostat I.V.), available since 1979, has been shown to be effective against *C. immitis, Candida* species, *Paracoccidicides brasiliensis,* and *C. neoformans,* but treatment failures have been reported commonly. The appropriate role for miconazole for other systemic fungal infections is poorly defined. Miconazole should not be considered the drug of choice for any systemic infection, but considered only in patients with treatment failure or relapses after amphotericin-B.

Miconazole is administered intravenously because it is poorly absorbed from the gastrointestinal tract after oral administration. Dosages are 400-1200 mg intravenously every eight hours for 8-12 weeks. It is metabolized by the liver and adjustments for renal dysfunction are not necessary. Side effects are numerous ranging from fever, chills, phlebitis, and pruritis to more severe medical problems such as leukopenia, thrombocytopenia, hepatitis, anaphylaxis, and hyponatremia.

Ketoconazole. Ketoconazole (Nizoral) was released in 1981 for clinical use. Ketoconazole has been shown to be effective against superficial mycoses, in particular the resistant forms of chronic mucocutaneous candidiasis. Experience in treatment of systemic fungal infections with ketoconazole is still being accumulated. The most dramatic clinical responses have been in patients with paracoccidioidomycosis and histoplasmosis. Although it may eventually become the drug of choice in paracoccid-

ioidomycosis, its role in other systemic fungal infections is poorly understood. Relapses have been reported at very high rates in histoplasmosis and other fungal infections even after a year of therapy. Because of its poor cerebrospinal fluid penetration, central nervous system infections should not be treated with this drug. There have been no controlled trials comparing ketoconazole to amphotericin-B in the treatment of systemic fungal diseases with respect to short-term and long-term effects.

Ketoconazole is given orally because of excellent absorption from the gastrointestinal tract. Absorption is hindered by elevating the pH of the stomach. Thus, serum levels are much lower in patients given ketoconazole with antacids, cimetidine, or in patients with achlorhydria. Ketoconazole is metabolized by the liver with little or no urinary excretion. Dosage adjustment is not necessary in renal failure. The usual daily dosage is 200-400 mg, but doses up to 1200 mg/day have been used by some investigators. There have been recent reports of gynecomastia in males and fulminant hepatic failure possibly induced by ketoconazole. In view of these reports, ketoconazole may not be the innocuous drug it was once believed to be.

Combination Therapy

In humans, there are anecdotal reports of combination chemotherapy for various fungal infections. However, there are very few controlled trials. A recent randomized trial compared amphotericin-B alone *vs* amphotericin-B plus 5-fluorocytosine in cryptococcal meningitis. Amphotericin-B at 0.4 mg/kg/day was given to 24 patients for 70 days. Amphotericin-B, 0.3 mg/kg/day, plus 5-fluorocytosine 150 mg/kg/day were administered to 21 patients for 42 days. The combination was associated with cure or improvement in more patients than amphotericin-B alone. Thus, amphotericin-B plus 5-fluorocytosine appears to be the treatment of choice for cryptococcal meningitis.

Little is known about other drug combinations. Miconazole appears to reduce the effectiveness of amphotericin-B because of interference with the binding of amphotericin-B to sterols. Ketoconazole has been reported to be both synergistic and antagonistic when given with amphotericin-B. Rifampin has been used with amphotericin-B to treat *Aspergillus* infections with good success but the number of patients treated is too small to recommend it as the combination of choice in these infections.

Summary

The number of drugs available for the treatment of systemic fungal infections is limited. Amphotericin-B is still the drug of choice for most systemic fungal infections and the standard to which all other drugs should be compared. 5-fluorocytosine has a narrow spectrum and only a few indications. Miconazole showed early promise but a high relapse rate has put it in a secondary role. Ketoconazole is the newest antifungal agent and can be given orally. Early reports of successful treatments with few side effects have been tempered by reports of significant relapse rates and possible serious side effects.

Self-Assessment Questions

1. Amphotericin-B should always be suspended in a 5% dextrose solution. (T or F)
2. Anemia that is secondary to amphotericin-B usually responds well to iron therapy. (T or F)
3. Potassium supplementation is necessary in about 25 per cent of patients receiving amphotericin-B. (T or F)
4. 5-fluorocytosine has a broad spectrum of activity against all systemic mycoses. (T or F)
5. Ketoconazole is very useful in treating meningitis and urinary tract infection. (T or F)
6. There are few indications at this time for combined therapy against systemic mycoses. (T or F)

(Answers on page 82)

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Drug INFORMATION

JOYCE GENERALI, R.Ph., *Editor*

Drugs That Increase the Serum Concentration of Digoxin

Digoxin is one of the most commonly prescribed drugs for the treatment of cardiac disorders. Due to the common use of digoxin, and multiple drug treatment required for some cardiac disorders, digoxin is commonly administered concurrently with other drugs. Thus there is the possibility of drug interactions involving digoxin. Such drug interactions can lead to decreases, as well as increases in the serum concentration of digoxin. The following discussion will focus on drug interactions that increase the serum concentration of digoxin.

Quinidine

Interaction: Several investigators have reported increases in serum digoxin concentrations in patients who are administered quinidine and digoxin concurrently.[1-6] Although the magnitude of the elevation in serum digoxin concentrations varies from 10 per cent to two-fold, some increase in the concentration is predictable. The mechanism of the quinidine-digoxin interaction is not fully understood, but those proposed include: (1) A reduction in the renal clearance of digoxin secondary to a decrease in the renal tubular secretion of digoxin;[1-8] (2) A reduction in the non-renal clearance of digoxin[9, 10] (especially important in patients with impaired renal function); and (3) A reduction in the volume of distribution of digoxin by displacing the drug from tissue binding sites.[1]

This interaction appears to be somewhat dependent on quinidine doses. Quinidine doses of 400-500 mg/day are reported to increase the serum concentration of digoxin approximately 50 per cent, while doses of 1000-1200 mg are reported to increase digoxin levels approximately 100 per cent or more.[6, 8, 10, 11]

Conclusion: Most patients treated concurrently with digoxin and quinidine will experience increases in the serum concentrations of digoxin. Although the magnitude of this increase will vary among patients, quinidine-induced digoxin toxicity has been reported in several patients.[3, 4] Patients should be monitored closely until the digoxin steady-state level is obtained during concurrent administration of these agents. Some authors suggest a 50 per cent reduction in digoxin doses during concurrent administration with quinidine to decrease the likelihood of digoxin toxicity.[4, 11]

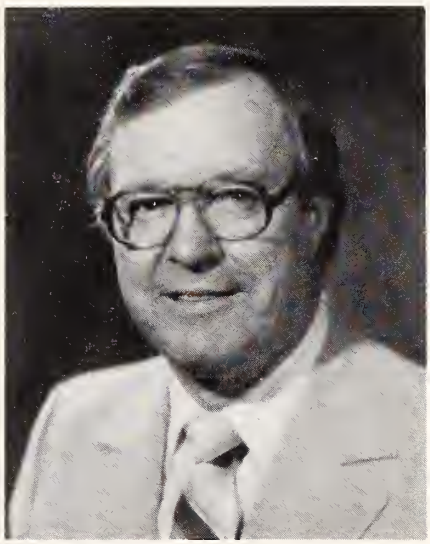
Quinine

Interaction: In 1981, Aronson and Carver reported an average increase of 43 per cent in the digoxin serum concentrations of four healthy volunteers treated with quinine sulfate, 1200 mg daily, after steady-state digoxin serum concentrations were obtained.[12] These investigators also reported a decrease in digoxin renal clearance from an average 2.32 to 1.86 ml/min/kg during administration of quinine. In 1980, Wandell *et al.* reported similar increases in serum digoxin concentrations following concurrent administration of digoxin and quinine.[13] However Wandell reported no change in the pre- and post-digoxin renal clearance or volume of distribution.[13]

Conclusion: Quinine appears to increase the serum concentration of digoxin when administered concurrently. Although the magnitude and mechanism of this interaction need further investigation, clinicians should be aware of the possibility of quinine-induced digoxin toxicity.

(Continued on page 80)

The President's Message



Dear Colleague:

The cost of health care will be the major issue in 1983. The people in power have determined that 10 per cent of the gross national product is all that we can allot to the health care industry. Accessibility and quality are no longer of major concern.

Since the inception of government programs, there has been a tendency to shift the cost of these programs to the private insurance industry. This shifting has reached the point where the private sector can no longer make up the difference without exceeding the affordable cost to business, labor, and private individuals. It appears that the more physicians were involved in third party carrier programs, the higher the benefits to physicians and the more "unaffordable" these plans have become to the average business and individual.

It would appear to me that we need to make significant decisions in these areas if we are to save our present system.

First dollar coverage is no longer an option. Deductibles of some magnitude will be necessary to decrease utilization. This comes as a disappointment to labor and many Americans, but is salable under present economic conditions.

A change in reimbursement to physicians and to hospitals is also essential, with some cap on expenditures unavoidable. This is a disappointment to hospital administrators and physicians alike. Some evaluation of essential *vs* nonessential care may also be necessary — a very difficult problem with our expanding technology and aging population.

The American Way has risen out of adversity. I am sure that we will face and resolve current problems in a manner that will protect our patients' health and still maintain the reputation of our profession.

President



Mind Your Words

Our ruminations last month about the risks inherent in medical practice deliberately stopped short of considering one which, while seemingly less threatening in its immediate effect, is potentially greater because of its ubiquitous nature. This danger is, in a word, words. This may sound as if we are going to bring up that unfortunately hackneyed term, communication, which we are but in its most fundamental sense since, without words, it is nothing. From the days of grunts and clicks (not entirely bred out of us), these sounds and their translations into scratches on clay or inked pictures on papyrus or even green characters dancing on a screen have been extended and shaped with increasing specificity which has at once simplified and complicated our means of passing around our thoughts. And because of the nature of medical communications, they are in no instance more important than in the medical world.

An example was noted in a recent issue of the *American Journal of Medical Genetics* where Laurence Karp reported editorially on the experience of a geneticist (probably himself although he skirts the identity) who was talking to a support group of parents who were bonded by the common grief of having defective children. In the course of his remarks, the geneticist made a comment about "gross defects" and was immediately taken to task by the chairman for insensitivity and offense against these people who were seeking to cope with (for many) an almost intolerable problem. It took him a moment to realize that his use of a perfectly legitimate, even essential, medical term had been heard, in this instance, in a derogatory sense. And it is probable that the incident, however innocent socially and appropriate professionally, affected the whole tenor of the occasion.

It must be one of the truisms of medical practice that, armed though he or she may be with impeccable academic qualifications, the physician is indoctrinated into the realities of medical service by a liberal education in the use of words by his patients — not just their lay meanings but the implications of medical authority which they carry. But under the exigencies of practice, the physician may neglect the process of attending closely to words — their professional meaning, yes, but what of their personal meaning to patients? It is less a matter of quantity than quality, and words can often rank with more evident therapies in importance (and not just in psychiatry).

There is no more consistent complaint by patients than their insistence that they do not get an adequate portion of the physician's time and attention. Whatever legitimacy is bestowed on such complaints by pressures or personalities, we submit that they stem not so much from the physician's neglect of duty as failure to achieve that essential contact with the patient by the judicious use of appropriate words. A distressing, even dangerous, result may be read in the patient's sometime preference for the dubious medical expertise of friends or the media and it has been demonstrated for centuries that the charlatans know, if nothing else, the power of their words on the sick.

This may seem to advocate the attitude that has been expressed in some quarters that the language should be subverted (and taught) at the usage level of the listeners. This is by no means our intent. The unhappy social defects will not be rectified by making the deficiencies the standard. The physician should be the medium of education in the medical field lest the patient's inability to grasp the medical purpose of the words be perpetuated. And the process begins when the physician learns to translate the medical terms into an effective exchange without prostituting them by condescension or dismissal — mindful that the listener, however versed in medical terms, is listening through a filter of intense self-concern. One need only consider for a moment the impact of the word *cancer* to see this at work.

At the same time, there can be a specificity of

hearing which, regardless of the presentation, can refuse to admit that which is unwanted. The patient who fails to comply with instructions or later denies having received them or getting adequate consideration may be acting out this fear-based rejection of competent medical advice. Any physician's journal is replete with such examples, recognized or not, but it is not only as an exercise in semantic gymnastics or communicative rapport that they warrant consideration. Lawsuits have been based on the choice or delivery of words that were incorrectly interpreted by the patient — or injudiciously chosen by the physician. And there can be no doubt that a major factor in the estrangement between physicians and lawyers stems in no small part from the difference in their use of words. This is well known to anyone who has read with growing dismay or anger the particulars of professional deficiency in a notice that a suit has been instigated — and observed the casual manner in which lawyers consider such words no more than their standard communication.

The problems associated with our insistence on verbal communication with each other are not only the shadings and nuances of meaning produced by the social or ethnic or cultural or professional usage but the architecture that dictates their assembly for various purposes. Our particular concern, quite naturally, is the production of the medical article. While our own publication abides by the customs of the day, we admit (with the sincere hope that we do not alienate any of our actual or potential contributors) that we feel there is room for improvement in the forms of presentation of medical information which have evolved over the years. Certainly, honesty, accuracy, and completeness must be at the foundation, and the substance must not be distorted by ambiguity or equivocation. Satisfying these requirements can very possibly leave us with a simple tabulation of the elements of intent, method, findings and conclusions, but this can be as appetizing as, for example, eating the various ingredients of a cake (preferably chocolate) individually as compared with the delectable whole that can result when they are properly combined. The medical cookbook, like its kitchen counterpart, can delight chiefly by the image of the results.

The current custom admits of no interjection of personal opinion or leavening comment which can give the work distinctive personality. The result, of course, may make an unassailably suitable item for storage in the medical archives and sought out by subsequent authors for a proper place in their bibliographies. Authors, however, well know (thanks to oppressive editors) that an overriding factor in the construction of the paper must be its ability to withstand the critical eyes of detractors. This is not to condemn the system — it is dictated by the current investigative philosophies and seems at the moment to be the appropriate way to assure objectivity and validity by which the information will stand the test of time and experience. Furthermore, it can be argued that medical writing is not competitive with lay literary productions and should not be judged by anything like the same standards. Still it is regrettable that only by inference do we get a glimpse of what really went on in the process. What were the background stories about who really did the work? The order of the authors' names? The intolerable itch of the author to find out some truth as related to the departmental requirement that something had to get published?

Obviously, we have peeked into a Pandora's box which is better left closed lest we wind up with the traditional editorial overcoat of tar and feathers. Still we can't help thinking about our favorite medical article of all time. We have reported on it before but it bears repetition. It was a contribution to the *Transactions of the Kansas Medical Society* for 1867 by Dr. John W. Parsons of what was then Mt. Pleasant, Kansas (and we have no idea of what it is called now or if it still exists). The whole thing is but a few lines, but the first sentence will suffice to make the point: "Old P. quarreled with his family, procured three drachms of arsenic, put it into a milk can and left the country." At that point, Dr. Parsons states briefly the degree to which the various family members suffered from the act, the methods he used to treat them, and his opinion as to the most efficacious treatment — which, incidentally, was sufficiently effective that all survived. We grant that today the original paper would probably be immediately discarded in virtually any medical editorial office (including, probably, this one), but we also submit that Dr. Parsons has told us more about the incident, the victims — and himself — than can be conveyed by the stylized forms of today (although we *are* left to wonder whatever happened to old P. — as well as Mt. Pleasant).

But the medical risks must be viewed as an outgrowth of the liberties constantly taken with words. (As Mr. Browning was careful not to say — a man's reach must exceed his grasp, or what's a metaphor?) Words have a way of wandering about — within the professions, through various aggregates of society, acquiring new meanings and color and tone — some gaining favor to the level of nausea, others losing ground and being dismissed in the dictionaries as

(Continued on page 88)

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Drug Information

(Continued from page 76)

Calcium Antagonists

Interaction: In 1981, Belz *et al.* reported increases of 77 and 45 per cent in digoxin steady-state concentrations following concurrent administration of verapamil, 240 mg daily and nifedipine, 30 mg daily, respectively.[14] This study supports the observations of Klein who reported a 60 per cent increase in digoxin serum concentrations following concurrent administration of verapamil and digoxin.[15] In 1981, Pedersen *et al.* reported verapamil to decrease the renal clearance and non-renal clearance of digoxin by 20 and 60 per cent, respectively.[16] Pedersen *et al.* also reported a 23 per cent decrease in the apparent volume of the central compartment.[16] Thus there are multiple mechanisms of this interaction.

Conclusion: Both verapamil and nifedipine appear to increase the serum concentration of digoxin in some patients by 60-70 per cent. Such an increase in the serum concentration of digoxin can potentially lead to digoxin toxicity. Thus patients administered digoxin and verapamil or nifedipine concurrently should be monitored closely until the new steady-state concentration of digoxin is obtained. There are no reports of a drug interaction involving digoxin and diltiazem.

Antibiotics

Interaction: Approximately 10 per cent of patients excrete a significant amount of orally administered digoxin as cardioinactive metabolites.[17, 18] Although the exact mechanism of this conversion is unknown, the gastrointestinal flora appear to play an important role in the conversion of orally administered digoxin. Lindenbaum *et al.* found as much as a two-fold increase in serum digoxin concentration, and a considerable reduction in the elimination of cardioinactive metabolites, following a five-day course of erythromycin or tetracycline in three volunteers known to be high excretors of cardioactive digoxin metabolites.[17]

Conclusion: Although a relatively small number of patients are suspected of this interaction, a two-fold increase in the digoxin serum concentration can be dangerous. Thus clinicians should be aware of such an interaction when treating patients with digoxin and antibiotics concurrently. This interaction also appears to be potentiated by the administration of oral products having poor bioavailability. Thus such products should be avoided when this interaction is suspected.

Spironolactone

Interaction: Radioimmunoassay, utilizing a specific antibody with an affinity for digoxin, is a common method of determining digoxin serum concentrations. In 1977, Lichey *et al.* reported an average increase in the measured digoxin concentration of 181 per cent using the radioimmunoassay method following five days of concurrent digoxin and spironolactone therapy.[19] Spironolactone and its metabolites are structurally similar to digoxin causing them to have an affinity for the antibody used during the radioimmunoassy of digoxin. Thus digoxin serum concentrations in patients receiving digoxin and spironolactone concurrently may be falsely elevated.

Spironolactone has also been shown to decrease the renal clearance of digoxin thus increasing its serum concentration.[20] Steiness reported an average increase from 0.8 to 1.0 ng/ml in the serum concentrations of 12 patients after spironolactone was added to their therapy.[20] The digoxin serum concentration of one patient (not included in the average concentrations listed above) increased from 1.0 to 3.5 ng/ml following the addition of spironolactone.

Conclusion: The spironolactone-digoxin drug interaction may be either a drug-laboratory or drug-drug interaction. The drug-laboratory interaction involving these agents will reflect an increase in the measured digoxin serum concentration which may not be true of the actual serum concentration. However spironolactone may decrease the renal clearance of digoxin thus increasing the digoxin serum concentration. Clinicians should be aware of both the drug-laboratory and drug-drug interaction involving spironolactone and digoxin. If one or both of these interactions is suspected, utilization of an assay method other than radioimmunoassay will be helpful in determining the nature of the interaction.

Submitted by
Bruce Scott, R.Ph.

References are available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

F.Y.I.—

The Drug Information page of the January *Journal* was devoted to isotretinoin (Accutane), a drug recently approved for treatment of severe recalcitrant cystic acne.

In the January 21 issue of *JAMA*, there is a letter from Frank W. Yoder, M.D., who participated in the National Institutes of Health clinical studies of this drug. Dr. Yoder warns that FDA approval was based on limited data, and that his continuing evaluation indicates a potential for insidious delayed toxic reactions. He urges extreme caution until the scope and magnitude of the toxicity of isotretinoin has been clarified.

The Drug Information Service is maintained by the Department of Pharmacy at the K.U. Medical Center to promote rational drug therapy. The Service is staffed by clinically oriented pharmacists with access to accurate, current, and unbiased information. Information is available free of charge to any health care practitioner involved with patient care. Contact:

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Systemic Fungal Infections

(Continued from page 75)

Answers

1. *True*
2. *False*
3. *True*
4. *False*
5. *False*
6. *True*

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Interstitial Cystitis

(Continued from page 71)

should definitely be presented to one suffering with severe interstitial cystitis.

Urinary diversion, with or without cystectomy, will nearly always relieve the symptoms of the disease. It is a last resort, however, and should rarely be necessary. As Walsh indicated, it should be regarded as the ultimate confession of a therapeutic failure.

In conclusion, interstitial cystitis is an enigmatic disease which, although histologically benign, is no doubt a malignant nuisance to the patient. The preponderance of evidence indicates an autoimmune etiology; however, it is likely that there are other contributing factors. The diagnosis remains one based almost entirely on symptoms and cystoscopic findings. Conservative treatment should begin with bladder distention under general anesthesia and probably include instillation of DMSO. Should surgical intervention be indicated, cystolysis, cecocystoplasty, and finally urinary diversion may be utilized.

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4. Taina E: Curr Med Res Opinion, 7:423-428, 1981.



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WARNINGS: Anaphylactoid reactions have occurred in patients hypersensitive to aspirin (see CONTRAINDICATIONS). Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Peptic ulceration, perforation, or gastrointestinal bleeding can end fatally, however, an association has not been established. Rufen should be given under close supervision to patients with a history of upper gastrointestinal tract disease, and only after consulting the ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be attempted. If Rufen must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

PRECAUTIONS: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If developed, discontinue Rufen and administer an ophthalmologic examination.

Fluid retention and edema have been associated with Rufen; caution should be used in patients with a history of cardiac decompensation.

Rufen can inhibit platelet aggregation and prolong bleeding time. Use with caution in patients with intrinsic coagulation defects and those taking anticoagulants.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy, this therapy should be tapered slowly when adding Rufen.

DRUG INTERACTION: *Coumarin-type anticoagulants.* The physician should be cautious when administering Rufen to patients on anticoagulants.

Aspirin. Concomitant use may decrease Rufen blood levels.

PREGNANCY AND NURSING MOTHERS: Rufen should not be taken during pregnancy nor by nursing mothers.

ADVERSE REACTIONS: Incidence greater than 1%. Gastrointestinal: The most frequent adverse reaction is gastrointestinal (4 to 16%). Includes nausea*, epigastric pain*, heartburn*, diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence). Central Nervous System: dizziness*, headache, nervousness. Dermatologic: rash* (including maculopapular type), pruritus. Special Senses: tinnitus. Metabolic: decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS). *Incidence 3% to 9%.

Incidence less than 1 in 100. Gastrointestinal: gastric or duodenal ulcer with bleeding and/or perforation, hemorrhage, melena. Central Nervous System: depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma. Dermatologic: vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome and alopecia. *Special Senses:* hearing loss, amblyopia (blurred and/or diminished vision, scotomata and/or changes in color vision) [see PRECAUTIONS]. Hematologic: neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs' positive), thrombocytopenia with or without purpura eosinophilia, decreases in hemoglobin and hematocrit. Cardiovascular: congestive heart failure in patients with marginal cardiac function, elevated blood pressure. Allergic: syndrome of abdominal pain, fever, chills, nausea and vomiting, anaphylaxis, bronchospasms (see CONTRAINDICATIONS). Renal: acute renal failure in patients with preexisting significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria. Miscellaneous: dry eyes and mouth, gingival ulcers, rhinitis.

Causal relationship unknown. Gastrointestinal: pancreatitis. Central Nervous System: paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri. Dermatologic: toxic epidermal necrolysis, photoallergic skin reactions. Special Senses: conjunctivitis, diplopia, optic neuritis. Hematologic: bleeding episodes. Allergic: serum sickness, lupus erythematosus syndrome, Henoch-Schonlein vasculitis. Endocrine: gynecomastia, hypoglycemia. Cardiovascular: arrhythmias (sinus tachycardia, bradycardia, and palpitations). Renal: renal papillary necrosis.

OVERDOSAGE: Acute overdosage, the stomach should be emptied. Rufen is acidic and excreted in the urine, alkaline diuresis may benefit.

DOSAGE AND ADMINISTRATION: Rheumatoid arthritis and osteoarthritis, including flareups of chronic disease: Suggested dosage 400 mg t.i.d. or q.i.d.

Dysmenorrhea: 400 mg every 4 hours as necessary.

Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for the relief of pain. *Do not exceed 2,400 mg per day.*

CAUTION: Federal law prohibits dispensing without prescription.

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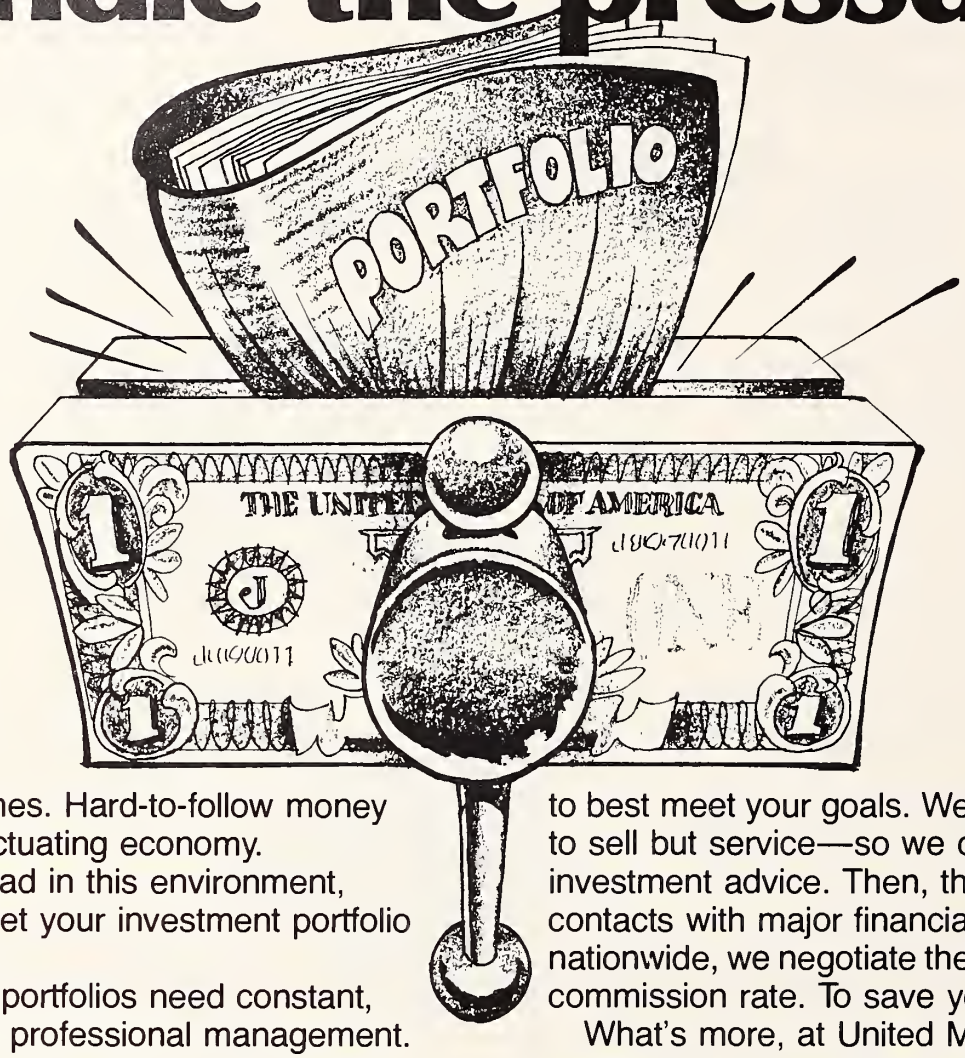
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Alcoholism, other drug abuse, and medical/neurological/ psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

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AMA House of Delegates

(Continued from page 47)

tussis immunization but, recognizing that there may be adverse effects with the current antigen, encourage development of improved vaccines.

Health Policy Agenda

The House considered a progress report on the major new AMA initiative called "Health Policy Agenda for the American People." The purpose of this project is to provide a broad-based outline of major concerns for the health sector. This project will give the AMA and others in the health sector (including various levels of government) the opportunity to abandon the traditional, year-by-year, piecemeal development of policy that responds only to pressures of the moment.

The Board reported that since the 1982 Annual Meeting in June, most activity has focused on:

- Establishing the various multi-organizational units that will actually conduct the analysis;
- Developing and refining the specific methodology that will be used in the initial phases of the work; and
- Organizing staff resources.

The project is expected to be of at least three years' duration.

The House also directed:

- That the AMA forward timely reports of the National Health Policy Agenda Steering Committee to all state medical societies and AMA delegates and alternates for review and input.
- That each of the six work groups be increased by two members who are in the private clinical practice of medicine and selected by the AMA president on a regional basis. Alex Scott, M.D., Junction City, was appointed to the AMA work group that will study payment mechanisms.
- That one additional individual be appointed to each work group by the Board of Trustees.

Vaccine-Related Injuries

The House called upon the AMA to support state and federal legislation that would hold physicians and manufacturers not liable for adverse reactions to immunizations required by law, providing there is no negligence.

By referral to the Board of Trustees, the House asked the AMA to consider supporting the development and enactment of national legislation establishing a compensation system for immunological agent-related injuries.

Diagnostic Related Groups

The House took the following action pertaining to the diagnostic related groups (DRG) prospective reimbursement system:

- That the Congress and the Department of Health and Human Services take an extremely cautious approach to this proposal;
- That the AMA study the proposal and submit a report to the House at the 1983 Annual Meeting;
- That the AMA petition the government to broaden its experimental reimbursement formula beyond the DRG concept; and
- That implementation be done on an experimental basis only and restricted to selected areas before being imposed nationally.

Conclusion

AMA House meetings provide a unique educational opportunity and we would encourage you to attend and participate. Any member of the Association may present testimony at the Reference Committee hearings. If you are unable to attend the meeting, you can still be represented through your delegates. Let us know your opinions. You can also prepare a resolution and request that it be submitted to the House. Many AMA policies began with an individual physician who had a good idea and coaxed it through the democratic process.

Thank you for giving us this opportunity to present this report.

Clair C. Conard, M.D., *Delegate*
Alex C. Scott, M.D., *Delegate*
Kermit G. Wedel, M.D., *Alternate*
Lew W. Purinton, M.D., *Alternate*

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Mind Your Words

(Continued from page 79)

arch. or *obs.* They are the instruments of inspiration for the evangelists — political, economic, religious — even health. They are weapons of the young against their elders and the fragile agents of the law, subjected constantly to scrutiny in search of true (at least, new) meanings and constantly requiring voluminous reinforcements or receiving ignominious dismissal.

All this produces an unease among those who work with words and see continuing threats to the world of words as we think we know it (even as we contribute to the changes). Such was expressed a few weeks ago by C. W. Guswelle, a columnist (and, we suspect, much more) with the *Kansas City Star,* who, in reporting his concern about the current uses and abuses of words, commented, "The larger question, however, may not be whether the public knows or cares but whether, just possibly, we have come unsuspecting to one of those fault lines in human events, usually detectable only in retrospect. Is this that moment in our culture when the competence of language, as we have known it, will one day be seen to have been lost — and with it so much else?"

Perhaps — or is it a matter of the slave becoming the master? And the pervading risk may be in our failure to recognize it. — *D.E.G.*

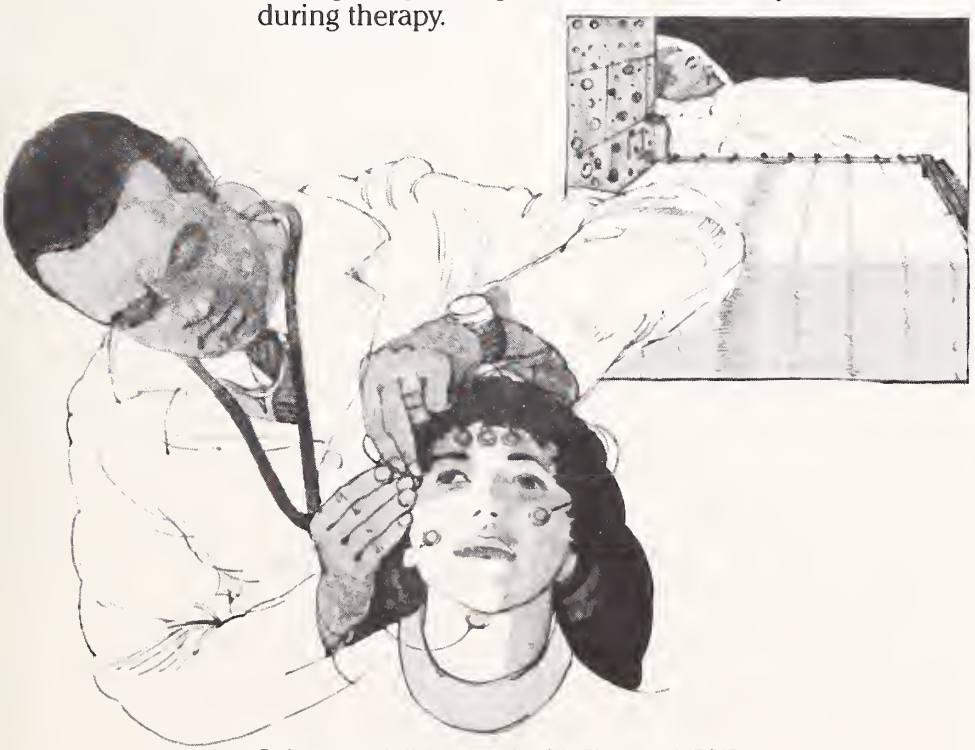
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Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, e.g., excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg recommended initially until response is determined.

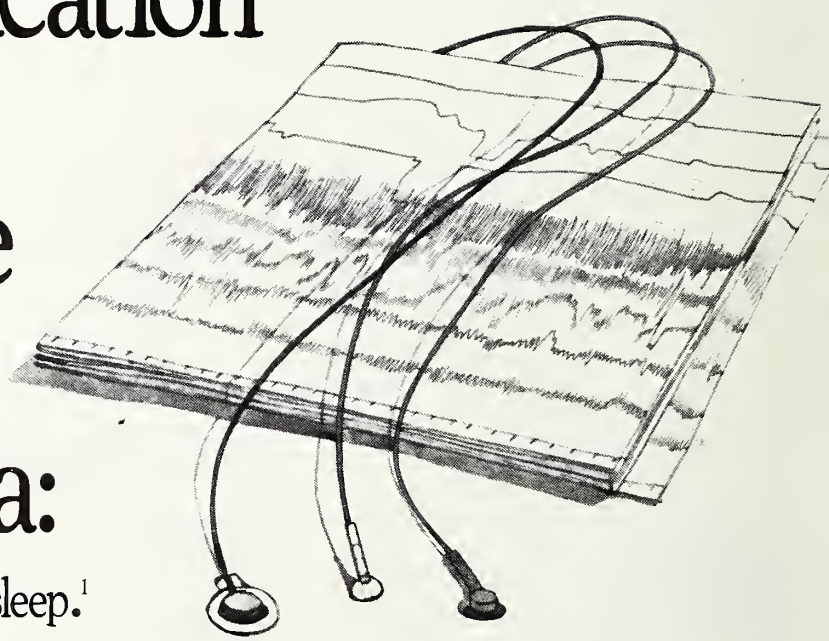
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Doctor, you may already be a WINNER . . .

You are if you've already registered for the **KMS State Meeting** to be held **May 5-8** at the **Holidome** in **Lawrence.**

But there's still time for you to win. . . . Pre-register by sending your check to the KMS office prior to April 13 for discounts on many of the following activities:

THURSDAY, MAY 5 — **SPORTS DAY**

8:45 a.m. to 1:15 p.m. — For the Athlete & Non-Athlete
— Welcome by KU Chancellor Gene Budig & Physical Education Chairman Wayne Osness
— Swimming, racquetball, tennis, aerobic dance, table tennis, badminton, jog on scenic tour of campus.
— Individualized physical fitness profiles ($15)
— CME Credit

Alvamar Country Club

11:00 a.m. — Golf
2:00 p.m. — Tennis (Doubles & mixed doubles)
2:00 p.m. — Trap Shoot

Sports Banquet

6:00 p.m. — Social Hour
7:00 p.m. — Dinner

FRIDAY, MAY 6

9:00 a.m. — First House of Delegates & Reference Committee
12:30 p.m. — KMS Delegates' Luncheon
2:00 p.m. — AMA/GTE Telenet (Computer System Program)
4:00 p.m. — KaMPAC Hospitality Suite
5:30 p.m. — Social Hour — KMS Auxiliary
6:30 p.m. — KMS Auxiliary Dinner followed by AMAERF Auction and Prairie Party

SATURDAY, MAY 7 — **SCIENTIFIC PROGRAM:** *Cancer in Kansas — 1983*

8:00 a.m. to 4:15 p.m. — Presentations will be by Stanley J. Vogel, M.D.; Richard Meidinger, M.D.; Jimmie Gleason, M.D.; George J. Farha, M.D.; Ronald L. Stephens, M.D.; Lawrence L. Tretbar, M.D.; T. S. Vats, M.D.; and James Bergin, M.D. The luncheon speaker, Howard N. Ward, M.D., will speak on "Euthanasia."
5:30 p.m. — Installation of new KMS President
6:30 p.m. — KMS President's Reception (Complimentary champagne & hors d'ouevres)

SUNDAY, MAY 8

7:00 a.m. — KMS Delegates' Breakfast
8:00 a.m. — Second House of Delegates
11:00 a.m. — KMS Council Meeting

For additional information, call the KMS office (in Kansas) at 800-332-0156
HURRY! Beat the deadline — April 13
BE A WINNER!

NOMINATING COMMITTEE

The Nominating Committee of the Kansas Medical Society, consisting of Herman W. Hiesterman, M.D., Chairman; Drs. Herbert Fransen, David M. Pugh, and Kermit G. Wedel, submits to the House of Delegates the following list of nominations for the elective offices of the Society. Wherever more than one nomination appears, they are presented in alphabetical order.

President

Jimmie A. Gleason, M.D., Topeka. Born in 1933. Graduated from the University of Kansas School of Medicine in 1958. Practices Obstetrics and Gynecology. Is now President Elect and has chaired the Legislative Committee for many years.

President Elect

F. Calvin Bigler, M.D., Garden City. Born in 1931. Graduated from Yale University School of Medicine in 1957. Practices General Surgery. Is Past President of the Kansas chapter, American College of Surgeons, and is now serving as First Vice President of KMS.

First Vice President

Clair C. Conard, M.D., Dodge City. Born in 1927. Graduated from the University of Kansas School of Medicine in 1955. Practices Internal Medicine. Now serves as AMA Delegate and has been Speaker of the KMS House of Delegates.

Second Vice President

Franklin G. Bichlmeier, M.D., Kansas City. Born in 1933. Graduated from the University of Kansas School of Medicine in 1958. Practices Cardiovascular Surgery. Is currently serving as Chairman of the Judicial Committee and as Vice Speaker.

David H. Clark, M.D., Salina. Born in 1936. Graduated from the University of Kansas School of Medicine in 1962. Is in Family Practice. Has served on the Continuing Medical Education Committee and is currently a member of the Mediserve Board.

Ivan E. Rhodes, M.D., Wichita. Born in 1925. Graduated from the University of Oklahoma School of Medicine in 1949. Practices Radiology. Has been a Councilor and is currently serving as Chairman of the Impaired Physician Committee.

Constitutional Secretary

K. William Bruner, Jr., M.D., Wichita. Born in 1944. Graduated from Harvard Medical School in 1970. Practices Pathology. Is active in KMS committee work and is currently serving as Constitutional Secretary and as Councilor.

Treasurer

Richard Meidinger, M.D., Topeka. Born in 1939. Graduated from the University of Kansas School of Medicine in 1965. Practices Diagnostic Roentgenology. Has served as Councilor.

G. Rex Stone, M.D., Manhattan. Born in 1929. Graduated from the University of Kansas School of Medicine in 1954. Practices General Surgery. Is now serving as Speaker.

Wayne O. Wallace, Jr., M.D., Atchison. Born in 1936. Graduated from the University of Missouri School of Medicine, Columbia, in 1965. Is in Family Practice. Is currently serving as Councilor and as Chairman of the Drug Utilization Review Committee.

Speaker

G. Rex Stone, M.D., Manhattan. Born in 1929. Graduated from the University of Kansas School of Medicine in 1954. Practices General Surgery. Is now serving as Speaker.

Vice Speaker

Franklin G. Bichlmeier, M.D., Kansas City. Born in 1933. Graduated from the University of Kansas School of Medicine in 1958. Practices Cardiovascular Surgery. Is currently serving as Chairman of the Judicial Committee and as Vice Speaker.

AMA Delegate

Lew W. Purinton, M.D., Wichita. Born in 1923. Graduated from the University of Kansas School of Medicine in 1948. Practices Internal Medicine. Is currently serving as AMA Alternate Delegate.

Kermit G. Wedel, M.D., Minneapolis. Born in 1932. Graduated from the University of Kansas School of Medicine in 1960. Is in Family Practice. Has served as Councilor. Is currently serving as AMA Alternate Delegate and as KMS President.

AMA Alternate Delegate

Ray E. Allen, M.D., Liberal. Born in 1937. Graduated from the University of Kansas School of Medicine in 1964. Practices Internal Medicine. Has served on the KMS Continuing Education Committee.

Jimmie A. Gleason, M.D., Topeka. Born in 1933. Graduated from the University of Kansas School of Medicine in 1958. Practices Obstetrics and Gynecology. Is now President Elect and has chaired the Legislative Committee for many years.

Robert P. Hudson, M.D., Kansas City. Born in 1926. Graduated from the University of Kansas School of Medicine in 1952. Practices Internal Medicine. Has long been active in KMS committee work.

Linda D. Warren, M.D., Hanover. Born in 1944. Graduated from the University of Kansas School of Medicine in 1970. Is in Family Practice. Is active in AMA and KMS committee work.

Join KaMPAC

dis·ease

/diz-ez/ 1. Chronic dependency upon alcohol; impairing the performance of vital functions. 2. Arrested through effective alcoholism treatment program.

Chances are, during a patient's preliminary examination, alcoholism is not one of your immediate suspicions. But with 10 million Americans suffering from this disease, and over 100,000 in the Kansas City area alone, perhaps it should be. Or perhaps the drinking problem is someone within your patient's family, causing them mental and even physical abuse.

At Bethany Medical Center's Alcoholism Rehabilitation Department (ARD), our professional staff offer both in- and outpatient programs for individuals suffering from alcoholism.

Our outpatient program is for those individuals whose affliction has not become debilitating, but recognize the need for treatment. It is specifically tailored to the individual's needs and incorporates family involvement.

Our inpatient program covers a 28-day phased treatment in a professional, yet unusual and cheery environment. Patients wear street clothes and participate in a busy, structured, daily routine that includes exercise, counseling, lectures and recreational time.

If you have a patient with a drinking problem, send for our detailed informational brochure, or personally contact one of our professional staff. Outpatient: Paul Anderson, Ph.D., Family Institute of Addiction Treatment, 7840 Washington Ave. Suite 300, K.C., KS. 66112. (913) 334-6300. Inpatient information: Sue Little, Director, Bethany Medical Center, 51 North 12th Street, K.C., KS. 66102. (913) 281-8951.

Alcoholism
Rehabilitation
Department

BMC

Bethany Medical Center

Swing-Bed Concept

The dramatic increase in the demand by older persons for hospital and health services is a critical factor affecting hospitals and physicians.

A recent questionnaire distributed at the Governor's Conference on Aging revealed that health care costs and the continued availability of health care are of grave concern to the elderly.

Drastic cuts in government supported programs, particularly in social services and in programs that affect those aged 65 years and older, may result in a shift to hospitals and physicians for care formerly provided by other health and social agencies. Changes in life-style and medical practice patterns may modify present utilization practices. Government regulations issued in 1982 allow small rural hospitals to implement the "swing-bed" concept of care. A swing-bed program in a rural hospital allows the patient to receive the necessary level of long-term care without being transferred out of the hospital or community.

Often it has been necessary to retain community residents as acute care patients in a community hospital beyond the time they should have been classified as long-term care patients because skilled nursing home beds were unavailable locally. Availability of swing-beds in rural hospitals will enable a patient to transfer to long-term care status at the appropriate time, thus reducing the cost to the patient while meeting a critical need — the patient's need to be close to family and friends.

Physicians are the principal agents in the referral and placement of patients. Please examine the positive features of the swing-bed approach. A swing-bed program may provide better continuity in medical care to the elderly and at a lower cost.

BRIEF SUMMARY
PROCARDIA® CAPSULES For Oral Use
(nifedipine)

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug Interactions: Beta-adrenergic blocking agents: (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

More detailed professional information available on request.

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Pfizer LABORATORIES DIVISION
PFIZER INC

"I can do things that I couldn't do for 3 yrs. including joining the human race again."



Quotes from an unsolicited letter received by Pfizer from an angina patient.
While this patient's experience is representative of many unsolicited comments received, not all patients will respond to Procardia nor will they all respond to the same degree.

© 1983, Pfizer Inc.

"My daily routine consisted of sitting in my chair trying to stay alive."

"My doctor switched me to PROCARDIA[*] *as soon as it became available. The change in my condition is remarkable."*

"I shop, cook and can plant flowers again."

"I have been able to do volunteer work...and feel needed and useful once again."

PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks, taking fewer nitroglycerin tablets, doing more, and being more productive once again.

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%).



for the varied faces of angina

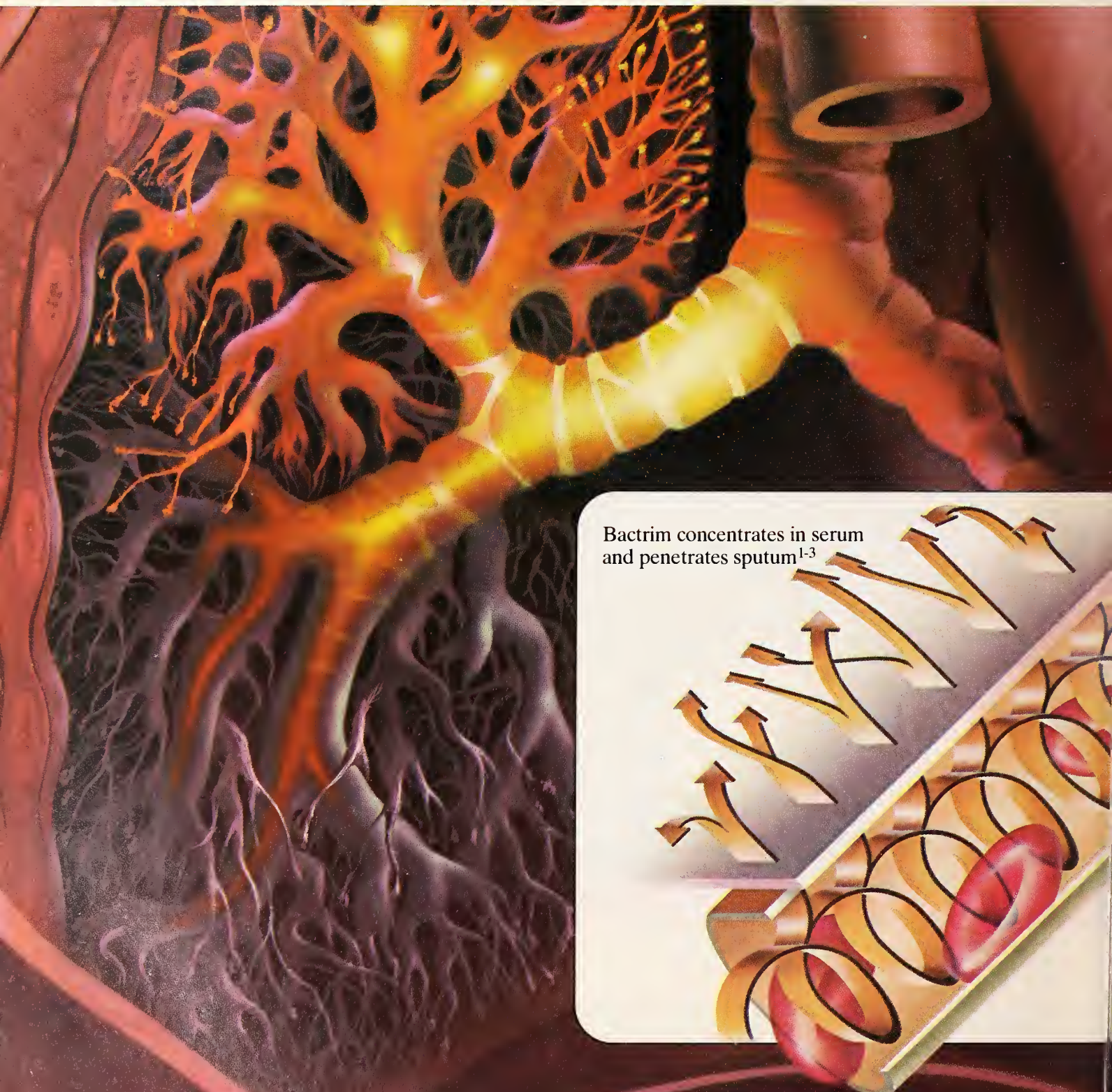
PROCARDIA®
(NIFEDIPINE) *Capsules 10 mg*

*Procardia is indicated for the management of:
1) Confirmed vasospastic angina.
2) Angina where the clinical presentation suggests a possible vasospastic component.
3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

Please see PROCARDIA *brief summary on adjoining page.*

Bactrim™ attacks the in acute exacerbations

(trimethoprim and sulfamethoxazole/Roche)



Bactrim concentrates in serum and penetrates sputum[1-3]

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ROCHE

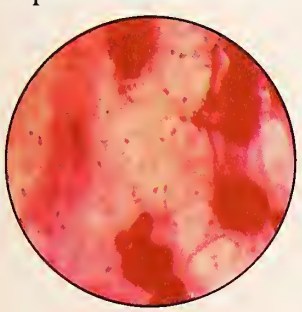
major pathogens of chronic bronchitis*

Bactrim clears sputum of susceptible bacteria

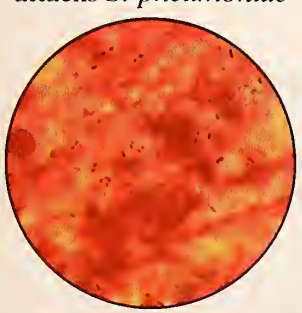
In sputum cultures from patients with acute exacerbations of chronic bronchitis, *H. influenzae* and *S. pneumoniae* are isolated more often than any other pathogens.[4,5] One study of transtracheal aspirates from 76 patients with acute exacerbations found that 80% of the isolates were of these two pathogens.[5]

Bactrim is effective *in vitro* against most strains of both *S. pneumoniae* and *H. influenzae*—even ampicillin-resistant strains. And in acute exacerbations of chronic bronchitis involving these two pathogens, sputum cultures taken seven days after a two-week course of therapy showed that Bactrim eradicated these bacteria in 91% (50 of 55) of the patients treated.[6]

attacks *H. influenzae*—even ampicillin-resistant strains



attacks *S. pneumoniae*



Bactrim reduces coughing and sputum production

In three double-blind comparisons with ampicillin *q.i.d.*, Bactrim DS proved equally effective on all clinical parameters.[7-9] Bactrim reduced the frequency and severity of coughing, reduced the amount of sputum produced and cleared the sputum of purulence.

Bactrim has the added advantages of *b.i.d.* dosage convenience and a lower incidence of diarrhea than with ampicillin, and it is useful in patients allergic to penicillins.

Bactrim also proved more effective than tetracyclines in 10 clinical trials involving nearly 700 patients.[10] Overall clinical condition of the patients, changes in sputum purulence, reduction in sputum volume and microbiological clearance of pathogens—all improved more with Bactrim therapy than with tetracyclines. G.I. side effects occurred in only 7% of patients treated with Bactrim compared with 12% of tetracycline-treated patients. (See Adverse Reactions in summary of product information on next page.)

Bactrim is contraindicated in pregnancy at term and nursing mothers, infants under two months of age, documented megaloblastic anemia due to folate deficiency and hypersensitivity.

Bactrim DS. For acute exacerbations of chronic bronchitis in adults* when it offers an advantage over single-agent antibacterials.

References: 1. Hughes DTD, Bye A, Hodder P: *Adv Antimicrob Antineoplastic Chemother I*/2:1105-1106, 1971. **2.** Jordan GW *et al: Can Med Assoc J 112*:91S-95S, Jun 14, 1975. **3.** Beck H, Pechere JC: *Prog Antimicrob Anticancer Chemother 1*:663-667, 1969. **4.** Quintiliani R: Microbiological and therapeutic considerations in exacerbations of chronic bronchitis, in *Chronic Bronchitis and Its Acute Exacerbations: Current Diagnostic and Therapeutic Concepts;* Princeton Junction, NJ, Communications Media for Education, Inc., 1980, pp. 9-12. **5.** Schreiner A *et al: Infection* 6(2):54-56, 1978. **6.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **7.** Chodosh S: Treatment of acute exacerbations of chronic bronchitis: results of a double-blind crossover clinical trial, in *Chronic Bronchitis and Its Acute Exacerbations: Current Diagnostic and Therapeutic Concepts. Op. cit.*, pp. 15-16. **8.** Chervinsky P: Double-blind clinical comparisons between trimethoprim-sulfamethoxazole (Bactrim™) and ampicillin in the treatment of bronchitic exacerbations. *Ibid.*, pp. 17-18. **9.** Dulfano MJ: Trimethoprim-sulfamethoxazole vs. ampicillin in the treatment of exacerbations of chronic bronchitis. *Ibid.*, pp. 19-20. **10.** Medici TC: Trimethoprim-sulfamethoxazole (Bactrim™) in treating acute exacerbations of chronic bronchitis: summary of European clinical experience. *Ibid.*, pp. 13-14.

Economical b.i.d.

Bactrim™ DS

(160 mg trimethoprim and 800 mg sulfamethoxazole/Roche)

*Due to susceptible organisms. **Please see next page for summary of product information.**

Bactrim™

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.
For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.
For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.
For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.
Also for the treatment of documented *Pneumocystis carinii* pneumonitis.
Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.
Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.
Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.
Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.
Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.
Dosage: Not recommended for infants less than two months of age.
URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:
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Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.
For patients with renal impairment. Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.
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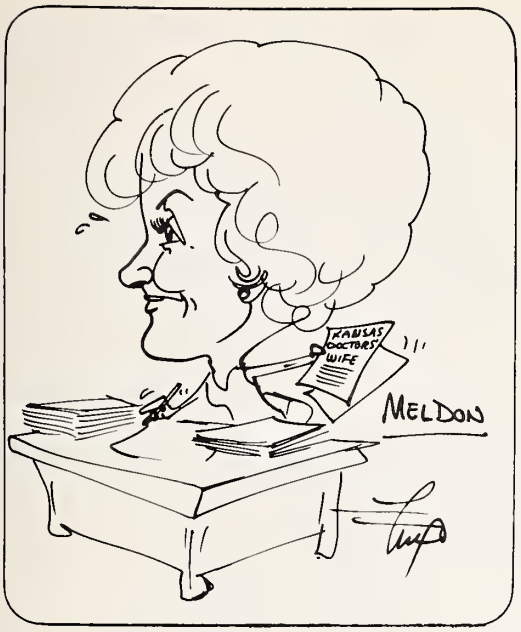


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AUXILIARY NEWS

An Open Letter to Kansas Physicians

Dear Doctor:

In a few weeks I will have earned membership in one of the most exclusive clubs in Kansas — the Past Presidents' Club of Kansas Medical Society Auxiliary. The entrance fee is a year of service as President of KMSA — plus a lot of blood, sweat, and tears! The past presidents have been the most supportive group of women I have ever known, and I shall do my best to carry on that tradition. I also have appreciated the support of the chairman of our advisory group, Dr. Emerson Yoder; the President of KMS, Dr. Kermit Wedel; and the staff of the Kansas Medical Society.

My duties as president included planning the programs for the Summer Conference in Ottawa, Mid-Winter Conference in Topeka, and State Convention in Lawrence. I attended two national meetings in Chicago, a conference on Drug Abuse in Kansas City, and will also attend the KMS "Wellness *vs* Abuse" conference in Salina in April. It has been a privilege to attend the KMS Board meetings and to know the physicians who so competently conduct the business of KMS.

Our main focus this year has been on drug abuse. We have taken our "Fonz and Friends" puppet show to many fourth and fifth graders, and our efforts to convince them of the dangers of marijuana were rewarded with their enthusiastic response. We have shown films on drug abuse, have sold STOP POT bumper stickers, and printed 3,000 "Dirty Dozen" brochures about drug abuse for our members to distribute.

Last year more than $21,000 was raised for AMAERF in Kansas. All states together and contributions by the AMA totaled $2 million. Of that, $1,700,000 was raised by the Auxiliary. We will have another auction at Convention, and we hope you will be there to bid!

This has been the busiest year of my life — yet a very rewarding one. The accomplishments of our county auxiliaries have given me material I will be proud to report to the AMA Auxiliary convention in June. The greatest desire of KMSA is to be an asset to you, both personally and as an organization, and I hope we have accomplished that this year.

Thank you for the opportunity to tell the Auxiliary story in this column.

Meldon Laury
President
Kansas Medical Society Auxiliary

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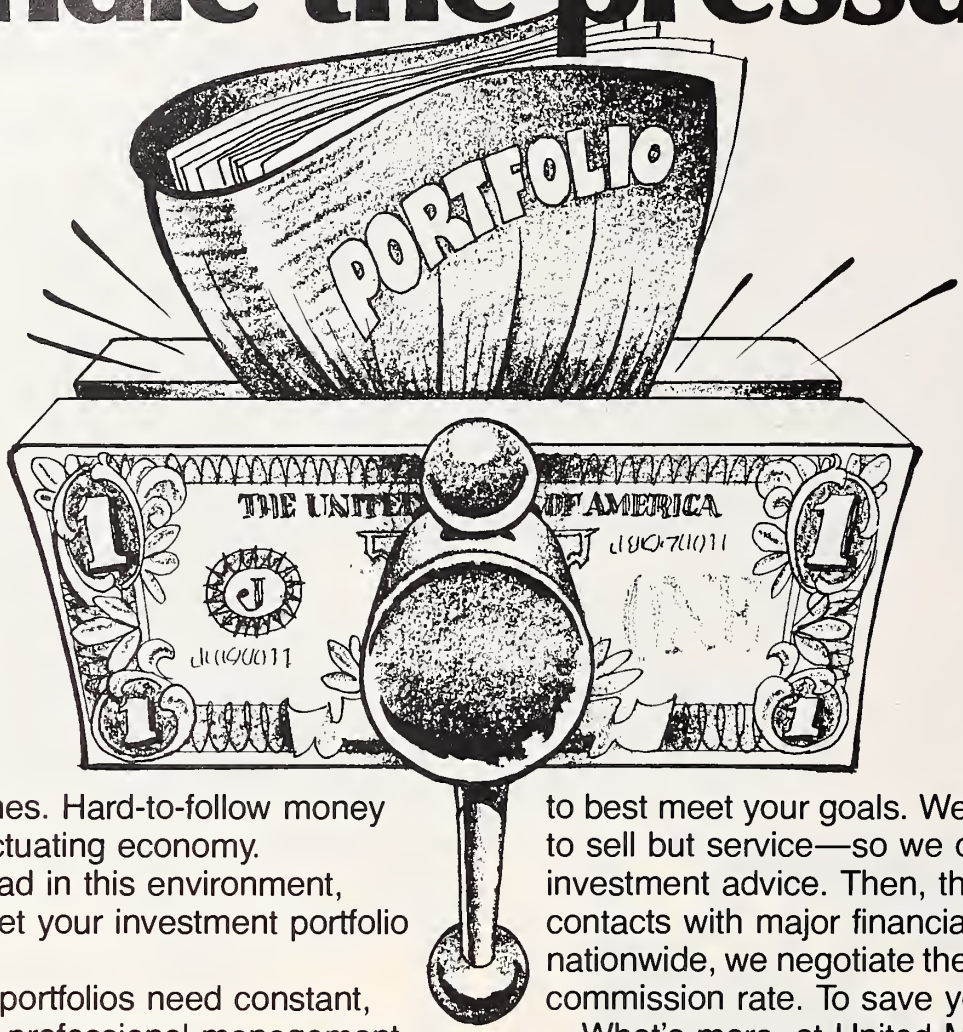
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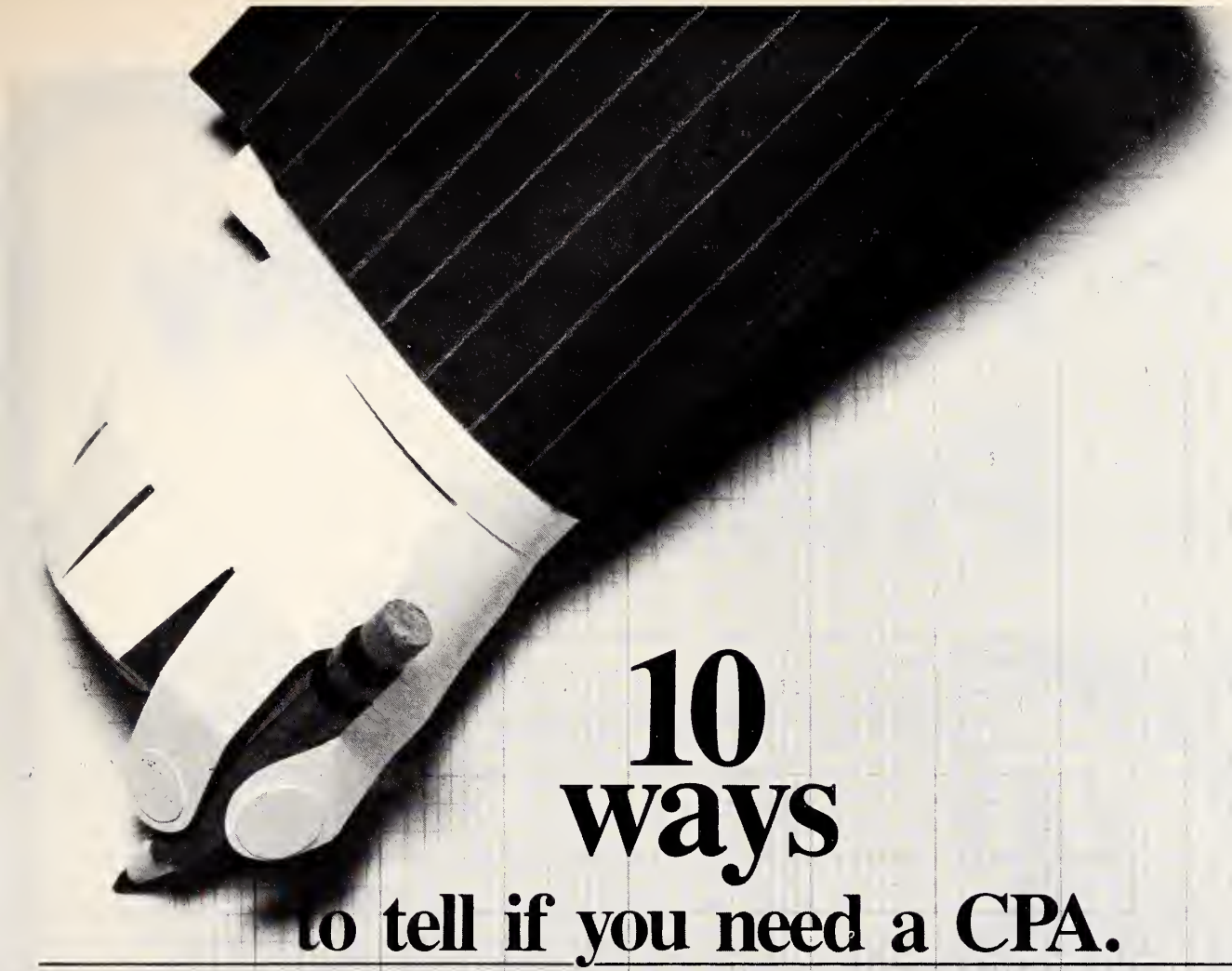
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Thirty-Seventh Annual University of Kansas School of Medicine Issue

The Dean's Letter — 1983

The University of Kansas School of Medicine-Kansas City

MARVIN DUNN, M.D.,* *Kansas City, Kansas*

At the completion of my second year as Dean of the University of Kansas School of Medicine-Kansas City, I am pleased to share with you the past year's accomplishments of your medical school. I hope you will be pleased, as I am, with these achievements.

We have attracted 800 students of high academic caliber to our medical school — 700 in Kansas City and 100 in Wichita. In keeping with our six-year record, our incoming freshman class had a grade point average of 3.6 and Medical College Aptitude Test (MCAT) average of 9.6 — the 65th percentile of all students taking the MCAT examination. These students have maintained their academic excellence by scoring above the 50th percentile for all medical students who have taken Part I (basic science portion) of the National Board Examination required by nearly all medical schools.

More than 80 per cent of our students match for their first or second choice in the Intern Matching Program. About one-half stay in this area for residency training; the remainder go elsewhere.

At the conclusion of residency training, most remain in this area to practice. Health Manpower records show that more than 50 per cent of Kansas physicians in primary care disciplines are graduates of UKSM and its residency programs. More than 60 per cent in family practice or general practice are UKSM graduates. Thus our medical school is fulfilling its obligation to provide physicians for the people of Kansas.

* Dean, University of Kansas School of Medicine-Kansas City.

A postgraduate International Symposium on Ventricular Arrhythmia, partially subsidized and co-sponsored by Marion Laboratories, brought scientists from many countries to our campus.

Remodeling has been completed at the Ophthalmology and Otorhinolaryngology clinics and offices. A non-invasive cardiovascular laboratory, equipped with a new computerized electrocardiography system, has also been completed.

Faculty size has remained stable, and future replacements will be primarily for retiring or migrating personnel. Our research scientists have received more than $12 million in research grant support. Our faculty research produced 1250 publications, and more than 1200 presentations were made before scientific societies.

Fifteen UKSM faculty members serve on scientific study committees of the National Institutes of Health, the National Cancer Institute, and the National Science Foundation. A number are on editorial boards of scientific journals and still others serve as editorial consultants.

The new Health Sciences Library is progressing on schedule with construction scheduled for completion in March and occupancy in May.

Our major challenge will be to maintain the quality of our educational, research, and service programs in the presence of a recessionary economy. Steps have been taken to consolidate programs and reduce non-essential personnel to conserve resources. We appreciate your continued support of our efforts.

The Chancellor's Letter — 1983

The University of Kansas

GENE A. BUDIG, Ph.D.,* *Lawrence*



I AM GRATEFUL for this opportunity to share a few observations and concerns with the medical community of the State of Kansas. The vitality of our institutions of higher learning is directly affected by the continued economic malaise of the state and nation. Within this adverse climate, medical education and the University of Kansas Medical Center continue as top priorities for the University.

The challenge of UKSM to ensure a rich and varied educational experience for its students can be met only with a combination of three ingredients: motivated and responsible young people; effective faculty; and adequate resources. The supply of students seems assured; the other two factors are a source of concern.

The significant decline in physicians who choose to enter academic medicine is at least partially related to the sharp decline in research funds — a strong motivation to academically oriented physicians. Thus, we face the threat of a dwindling supply of articulate, committed physician-investigators to educate our medical students. No profession other than medicine better exemplifies the relationship between advances in human well-being and progress in scientific research. We must continue to attract new medical faculty and to retain those of our current faculty whose scientific, clinical, and educational abilities ensure a meaningful experience for our students. We must also do more to encourage those medical students and residents who are prospective academicians by creating an academic track that blends a special sequence of clinical and research experiences for interested students.

The appropriate education and training of future physicians requires exposure to a diverse and extensive mix of patients. There are tremendous societal, governmental, and economic factors working against the contemporary university hospital in this country. The University of Kansas Medical Center must compete for patients with private and other public hospitals, but the stakes in this competition are much higher than revenues; they bear upon the very quality of medical education itself. Clearly, our ability to provide appropriate clinical experience must be preserved. We must work more effectively with community physicians and hospitals throughout the state in order to develop a proper flow of patient referrals to the Bell Memorial Hospital. I am confident that our medical staff will work diligently with you toward that end.

With your help, UKSM will meet the challenges posed by the health care marketplace, and will protect those educational programs and scholarly activities that make it a unique resource, for ourselves and for future generations of health professionals.

* Chancellor, University of Kansas: Lawrence, Kansas City, Wichita.

The Vice Chancellor's Letter — 1983

The University of Kansas School of Medicine-Kansas City

JOSEPH C. MEEK, JR., M.D.,* *Kansas City, Kansas*

NEW CONSTRUCTION, new programs in graduate education, and strengthening and restructuring areas of clinical excellence mark the academic progress at the University of Kansas School of Medicine-Kansas City during the past year. Despite declining federal support for education of health professionals, we have continued to consolidate and improve our educational and research programs in the Schools of Medicine, Nursing, and Allied Health. Our Outreach program continues to expand in offering continuing education opportunities to the health professionals in Kansas, and with the tremendous support and cooperation of Kansas physicians, our two-month medical preceptorship has received wide acceptance. There is no doubt that the additional time that our students spend with community physicians adds a valuable and meaningful experience to their medical education.

During the past year, a major academic accomplishment at UKSM has been the creation of comprehensive medical programs which combine research and education with excellence of service. They include the following:

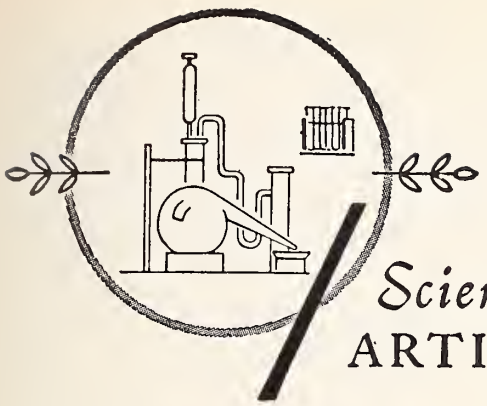
- Amyotrophic Lateral Sclerosis (ALS) Research Center at UKSM-KC and the Kansas City Veterans Administration Medical Center (Department of Neurology);
- Diabetes Clinical Research Center;
- International Center for the Control of Nutritional Anemia (funded by the U.S. Agency for International Development and the World Health Organization);
- Kidney and Urologic Disease Center;
- Comprehensive Cancer Center; and
- Center for the Study of Heart and Circulatory Disorders.

The Gerontology Center at UKSM is actively involved in a broad range of multidisciplinary academic and clinical programs. The Center supports research efforts and contributes to Outreach and Continuing Education programs as well.

The new Dykes Library for the Health Sciences, to be completed this year, will greatly improve the learning environment on the Kansas City campus. Expansion of library facilities has been a critical priority to faculty and friends, who have contributed more than $250,000 toward the new facility.

As exciting and positive as the academic accomplishments have been during the past year, new academic thrusts are taking place at this time. In response to the decline in the training of health professionals who pursue a career in academic medicine, our faculty are creating a new educational program for medical students at UKSM. An academic track program is being formulated that will support and encourage medical students to actively engage in biomedical research throughout their medical education, and that will promote their interest in an academically oriented medical career. This program will help to ensure the perpetuation of the superb clinical faculty that exists today at the University of Kansas Medical Center.

* Vice Chancellor for Academic Affairs, UKSM-KC.



Individualized Treatment

Regimen Design for the Problem Type II Diabetic

BRUCE E. JOHNSON, M.D.; JOSEPH L. KYNER, M.D. *and*
ROBERT E. BOLINGER, M.D., *Kansas City, Kansas*

RESISTANCE to the development of ketoacidosis in the absence of insulin administration is the key distinguishing feature of the noninsulin-dependent diabetic (Type II). Because of the absence of this minatory event, many Type II diabetics receive a late diagnosis, and if diagnosed, are allowed to continue in a state of chronic or asymptomatic hyperglycemia, using the absence of glycosuria as the only criterion of control.[1] An increasing body of data[2] implicates chronic hyperglycemia as an important causative factor in the development of the degenerative complications of diabetes and serves to emphasize the vital role of normoglycemia in their prevention. Longitudinal studies tend to identify a mean plasma glucose of 140 mg/dl as a point above which there is a correlation with an increased incidence of degenerative complications. Since mean plasma glucose of Type II diabetics among general medicine patients is between 200-300 mg/dl and Type II diabetes constitutes 80 per cent of all diabetics, this indicates a large group of patients at potential risk. In addition to hyperglycemia more comprehensive criteria of regulation must be considered, *i.e.* glycosylated hemoglobin[1] and high density lipoprotein (HDL) cholesterol. The prevalence of adiposity in this group is well known, and the effectiveness of dietary regulation is established as a means of attaining normoglycemia.

The application of available therapeutic modalities — *i.e.* diet, insulin, oral hypoglycemic agents, and exercise — to the Type II diabetic are often clinically applied in a haphazard manner. The development of an apparent *laissez-faire* attitude among both patients and health care personnel is engendered by the observation that, regardless of the type of intervention, the control parameters often remain in an unacceptable range. An approach to effective control, however, must begin with a recognition and assessment of factors that lead to the hyperglycemia in these patients.

The Type II diabetic is typically noninsulin dependent. In spite of a lack of glycosuria, such patients are often chronically hyperglycemic. Poor regulation may result from a number of factors, as illustrated by these three cases who successful treatments resulted in long term normoglycemia. In such patients, none of the usual therapeutic modalities is successful until insulin resistance is overcome.

Determinants of the Decompensated State

Regardless of whatever factors are operative in the determination of the state of glycemia, the crucial point is transmission of the extracellular insulin

From the Department of Medicine, The University of Kansas School of Medicine-Kansas City.
Address reprint requests to Dr. Bolinger, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.

signal to the intracellular mechanisms regulating glucose uptake by the cell. This is dependent on the following sequence:

1. The quantity and quality of endogenous insulin secretion;
2. The transport and fate of the insulin in the plasma;
3. The target cell resistance to insulin; and
4. The metabolism of the cell.

A number of hormonal as well as nonhormonal factors are known to influence these steps. These relationships are summarized in *Figure 1*. In the clinical management of the Type II diabetic, the physician is primarily concerned with the first three steps.

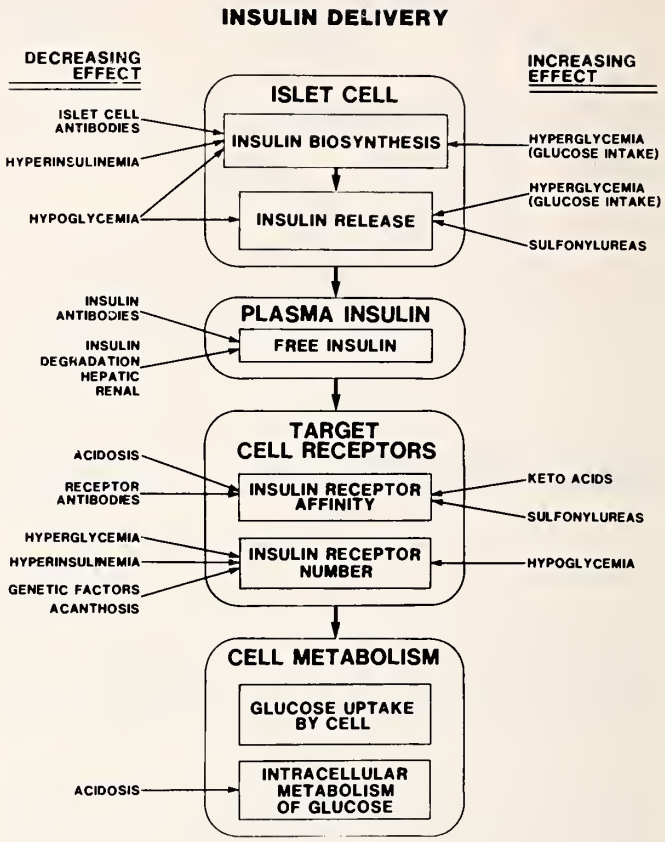


Figure 1. The four major steps from insulin formation to cellular response are shown. Factors that decrease or inhibit each step are shown on the left, and those that increase or facilitate, on the right.

Endogenous Insulin Secretion

In contrast to the Type I diabetic, the Type II diabetic characteristically shows evidence of endogenous insulin secretion. Measurement of plasma insulin levels (IRI) reveals normal or even elevated values in Type II diabetes. Many diabetics in this group are, or have been, on insulin, which makes determination of the IRI relatively useless because of the presence of circulating antibodies to insulin. Special techniques of insulin extraction can correct for this but are cumbersome to perform. The level of insulin C-peptide in the plasma is an accurate reflection of endogenous insulin secretion, since this peptide is secreted with insulin from the islets in a linear relationship but is not present in administered insulin. Furthermore, the assay is reliable and not skewed by presence of insulin antibodies. Basal C-peptide levels distinguish three groups of diabetics:[3]

C-peptide levels below 0.6 ng/ml are insulin dependent.

C-peptide levels above 1.1 ng/ml are usually noninsulin dependent.

C-peptide levels between 0.6-1.1 ng/ml are an indeterminant group.

Randell *et al.*[4] have noted that the Type II diabetic may undergo periods of decompensation, during which the C-peptide levels are very low but return to normal after regulation is effected. Thus, in the decompensated state the ultimate compensated capacity of the pancreas to secrete insulin cannot be predicted. When regulation is accomplished, a level of C-peptide above 6 ng/ml after a glucose challenge usually indicates insulin independence, whereas levels below this indicate some degree of dependence on exogenous insulin. In the unregulated hyperglycemic state, a high level of circulating insulin from exogenous sources may serve to suppress insulin secretion by the islet[5] and defeat the purpose of administering large doses of insulin to such patients *(Figure 1)*.

Originally, the sulfonylureas were thought to facilitate insulin secretion but more recent work minimizes this as a major mechanism for the hypoglycemic effect of these agents.[6]

Transport and Fate of Insulin

Normally, binding of insulin in the plasma exists only when insulin antibodies are present as a result of previous repeated exposure to exogenous insulin.[7] The functional characteristics of such antibodies are quite variable. In most patients no effect is noted, but in some patients the antibodies may reversibly bind with insulin to the extent that the insulin activity is prolonged, thus changing the dosage and timing of insulin injections.[8, 9] Such problems are most likely to be seen in the Type I diabetic. At the other extreme, antibodies may irreversibly bind insulin and even in the Type II diabetic produce

a picture of complete insulinoprivia. This can be overcome by escalation of insulin dosage to levels that are high enough to saturate the antibody pool or by changing to a different animal source for the insulin. In any case, the continued administration of an antigenic insulin tends to perpetuate the production of insulin antibodies. These antibodies may also act against any endogenously secreted insulin[10] causing the Type II diabetic to behave like a Type I. The event that sets off this chain of events is not always clear, but is commonly related to an anamnestic response to infection. Treatment of the infection and withdrawal of antigenic insulin will usually break the cycle.

Target Tissue Resistance to Insulin

A third more common factor contributing to hyperglycemia in the Type II diabetic is target cell resistance to insulin and may be noted even in the non-diabetic population. Of particular importance in this respect are the insulin receptors.[11] Receptor abnormalities have been demonstrated in adipose tissue, mononuclear cells, red cells, hepatocytes, and fibroblasts. The number of receptors on a cell is regulated by a balance between receptor formation (repletion) and destruction (depletion) through a process of endocytosis of receptors. Thus, the insulin receptor activity at any given time is a dynamic process regulated by a number of factors.[11]

Of particular importance is the receptor depleting effect of hyperglycemia and hyperinsulinemia.[12] These two factors are largely responsible for the insulin resistance of the obese person. This type of insulin resistance is especially amenable to therapeutic intervention since it can be reversed by normalizing blood glucose and insulin. The usual outpatient attempts to do this by prescription of a "physiologic" diet are notoriously unsuccessful except in rare instances. Davidson[13] has emphasized the use of an initial period of total fasting in the management

TABLE I
SUMMARY OF CASES

Case	*Sex/Age*	*Previous Insulin Treatment*	*Clinical Features*	*Pre-Treatment Blood Glucose mg/dl*	*Response to Intensive Hospital Treatment* *Initial*	*Followup*
1	M 62	15 U, 10 yrs 35 U, 1 yr	Diabetes 15 yrs; stable obese, orthostatic hypotension, C-peptide 2.2 ng/ml	500-600	Blood glucose 110 mg/dl in 6 days	Blood glucose 80-120 mg/dl, Ultralente 10 U, Chlorpropamide 250 mg
2	F 53	35 U, 7 yrs	Diabetes 17 yrs; obese, increasing	300-500	First hospitalization Blood glucose 300-500 mg/dl	Insulin dose increased to 45 U daily
	F 54	45 U, 1 yr	Continued weight gain hyperglycemia in spite of increased insulin	300-500	Second hospitalization (1 yr later) blood glucose 250 mg/dl	Blood glucose 100-120 mg/dl Chlorpropamide 250 mg, no insulin, 2 yr followup
3	F 70	15 U, 1½ yrs, increased to 120 U	Diabetes 10 yrs; slightly obese; pneumonia 6 mos prior to admission, escalating insulin dose and continued hyperglycemia. Serum insulin binding marked.	200-500	Blood glucose 110 mg/dl	Blood glucose 80-120 mg/dl during 2 yr followup; no insulin, no oral agents

of such patients. This can be effectively and safely carried out only in the hospital setting. Protein supplemented fasting accomplishes the same objective, serves better to maintain a favorable nitrogen balance,[14] and is better tolerated by the patient. Exogenous insulin should be withdrawn at the same time.

In addition to the number of receptors, the activity of the receptors influences the state of resistance. Olefsky and Reavan[15] have demonstrated that receptor activity is improved by sulfonylureas. Reavan and Dray[6] have presented evidence that this may be, indeed, the main component of the hypoglycemic effect of these compounds. Repletion of receptor function through regulation with sulfonylureas has been demonstrated by Eaton *et al.*[16]

Ketoacids have been shown to improve receptor activity[17, 18] while acidosis causes a down regulation *(Figure 1)*. Thus ketosis without acidosis may be an additional factor in reducing insulin resistance during fasting.

Receptor antibodies *(Figure 1)* may also impair receptor function.[6] This factor is not readily amenable to treatment and is frequently a component of an underlying autoimmune disease. Acanthosis nigricans has been associated with congenital insulin receptor defects.[19]

Therapeutic Considerations

The usual Type II decompensated diabetic encountered in practice is gaining weight and is chronically hyperglycemic (blood glucose greater than 200 mg/dl) in spite of moderate to high doses of insulin or sulfonylureas. In the absence of ketosis or other underlying disease processes, a basal insulin C-peptide level of 1.1 ng/ml or higher indicates endogenous insulin secretion, but in the decompensated state does not predict the ultimate secretory capacity of the islet cells. Scarlett *et al.*[20] indicate that such patients frequently have a combination of partial islet cell deficiency and insulin receptor resistance. With the proper application of common therapeutic modalities (diet, sulfonylureas, and insulin), the glycemia should be normalized and the underlying pathophysiologic mechanism revealed. The order of application of these therapeutic tools is crucial and the early attainment of normoglycemia is essential before further steps are taken. The multiplicity of problems of management is illustrated in the following three cases. Each of these patients initially presented as a moderately obese Type II diabetic who had chronic hyperglycemia in spite of treatment with insulin, oral hypoglycemics and "diet," and who was described as unregulated because of non-compliance. The response to intensive hospital management reveals that different problems existed in each case. Pertinent characteristics of each are summarized in *Table 1*.

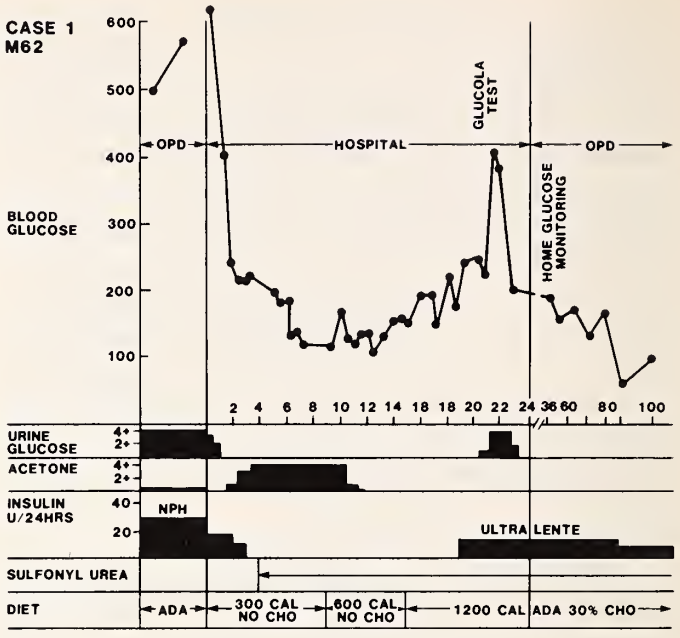


Figure 2. The response to treatment is shown for a Type II diabetic with elements of both insulin resistance and partial insulin deficiency.

Case One. The blood glucose in this patient responded promptly to carbohydrate deprivation indicating the absence of significant insulin blocking antibodies *(Figure 2)*. After normalization of the plasma glucose, an oral glucose challenge showed an insulin C-peptide response of 6 ng/ml indicating some insulin secretory deficiency. Ultimate regulation was optimal on 10 U of ultralente insulin and chlorpropamide, 250 mg. Thus the original problem was a combination of reversible receptor depletion and partial insulin deficiency.

Case Two. A summary of the first hospitalization of this patient is shown in *Figure 3*. At that time a 1500 calorie American Diabetic Association (ADA) diet was continued, insulin discontinued, and an oral hypoglycemic started. There was never normalization of blood glucose, and the patient was discharged on an even higher dose of insulin and continuing hyperglycemia.

A second hospitalization one year later is shown in *Figure 4*. A similar insulin withdrawal was carried out except that a complete fast was implemented. Blood glucose promptly responded, but climbed to unacceptable levels upon refeeding.

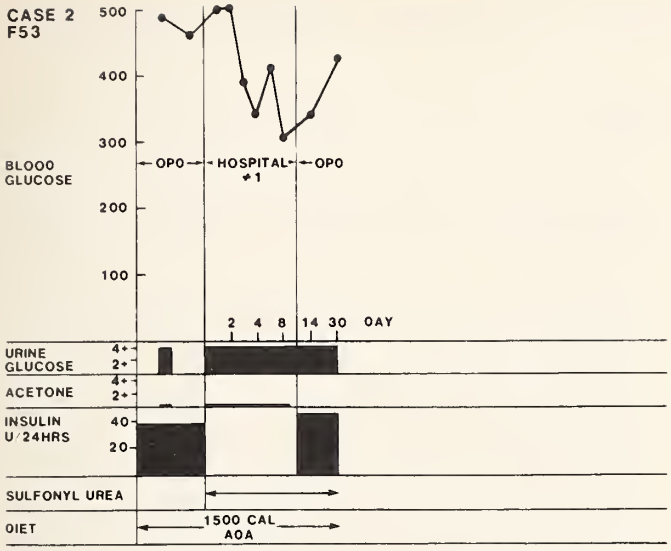


Figure 3. The poor response to treatment is shown for a Type II diabetic in which normoglycemia was never attained.

Upon addition of an oral hypoglycemic agent, blood glucose normalized. During a two-year followup, blood glucose has remained normal using the oral hypoglycemic agent without insulin.

In summary, the patient showed evidence of receptor depletion which was not corrected at the first hospitalization because of continued carbohydrate feeding but responded to the second attempt when carbohydrate was withdrawn and ketosis occurred. The addition of the sulfonylurea appeared to be a significant factor in maintaining normoglycemia without any insulin supplementation.

Case Three. A 70-year-old female had had diabetes diagnosed ten years prior to admission and had been treated intermittently with oral hypoglycemic agents. Two years before admission insulin was started at NPH 15 U daily. Six months before admission she developed pneumonia which responded to antibiotics. After recovery, though, the blood glucose ranged between 200-500 mg/dl in spite of escalation of insulin dosage to 120 U daily. The course in the hospital is shown in *Figure 5*. With caloric restriction and continuation of insulin, some improvement in glycemia was noted. Insulin was discontinued followed by some worsening of the glycemia. However, after five days plasma glucose returned to normal and has remained so during a two-year followup without insulin or oral hypoglycemic agents. Initially, the disappearance half time for tracer insulin was 110 minutes (normal eight minutes) indicating significant plasma binding of insulin. Serum electrophoresis with added tracer insulin showed significant insulin binding in the gamma globulin fraction. The amount of this binding decreased as soon as insulin dosage was discontinued. The rate of this decrease was similar to the expected rate of decrease of antibody globulin.

In summary, a significant factor in the insulin

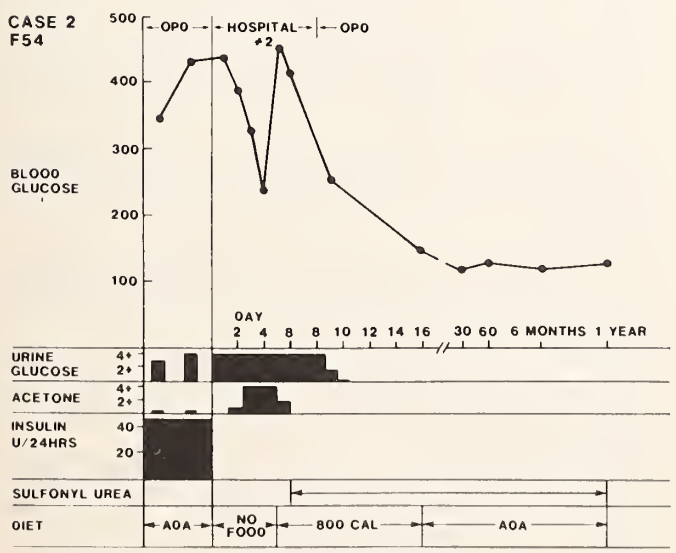


Figure 4. The favorable response to treatment is shown for the same diabetic when the regimen was initiated with total fasting.

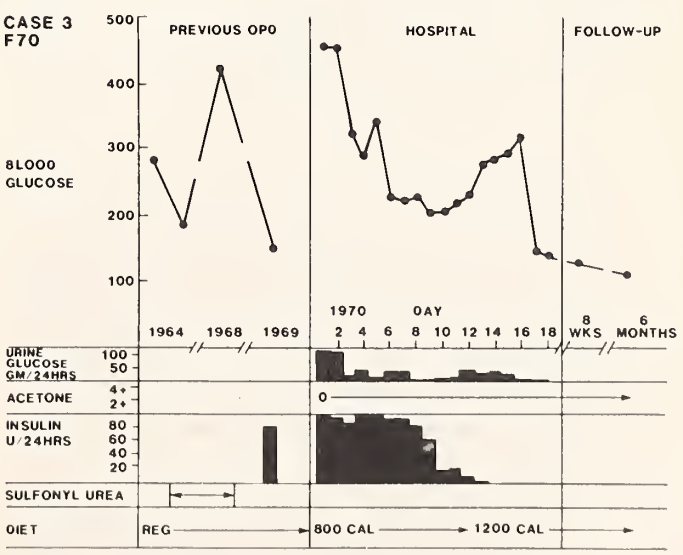


Figure 5. The response to treatment is shown for a Type II diabetic in which insulin resistance was due to antibody binding of insulin in the plasma.

resistance in this case was serum antibody binding of insulin. Upon discontinuation of exogenous insulin, a lag phase in glycemic response was noted corresponding to the time required for insulin antibodies to decrease, thus allowing the patient's endogenous insulin to become effective. The initial event causing decompensation was most likely an anamnestic antibody response to her infection.

Discussion

In the Type II diabetic, multiple factors may contribute to the state of decompensation *(Figure 1)*. Of importance among these are the endogenous insulin secretory capacity, serum antibody binding of insulin, and insulin receptor number and function at the target tissues. Although the determination of the insulin C-peptide may provide evidence for endogenous insulin secretory function, it cannot predict the ultimate secretory capacity. The following stepwise approach is recommended in the attempt to manage the decompensated Type II diabetic.

Insulin Receptor Repletion Regimen

1. Should be carried out in a hospital.
2. Withdraw insulin totally. If there is doubt about whether this is Type I or Type II diabetes, the withdrawal may be done over three days.
3. Withdraw all dietary carbohydrate and initiate a diet as follows: 300 calories, all protein and fat with an equal number of grams of each being most practical.
 Mineral supplements (Na, K, Ca) and vitamin supplements as needed.
 Large amounts of non-caloric fluids and bulk.
4. Monitor the following daily:
 a. Urine acetone: This should become positive in one to three days. If it does not, cheating should be suspected.
 b. Serum bicarbonate: As long as serum bicarbonate remains normal, the development of ketoacidosis is unlikely. If serum bicarbonate falls below 20 mEq/l, acidosis is likely and a significant insulin deficiency should be suspected. The regimen should be discontinued.
 c. Blood glucose should start to fall immediately, but other factors such as insulin antibodies may actually cause the glucose to rise for several days before a decline occurs. In some cases this may not occur for 10-12 days. Typically, the plasma glucose should go below 140 within three to five days. As long as acidosis or a hyperosmolar state does not ensue, the regimen may be continued.
5. As soon as insulin is discontinued, start an oral sulfonylurea.
6. As soon as normoglycemia appears, double the amount of protein and fat for one day.
7. If glucose remains normal, gradually increase to an isocaloric ADA diet but with 30 per cent carbohydrate, instead of the usual 40 per cent.
8. If the plasma glucose initially responds and then begins to climb to unacceptable levels when refeeding is begun, an element of endogenous insulin deficiency is likely. This may be confirmed at this time by the use of a two-hour oral glucose tolerance test. If the insulin C-peptide level does not rise to 6 ng/ml or greater, insulin deficiency is likely and small doses of ultralente insulin should be added to the above regimen to supplement the endogenous insulin secretion.

Although a number of tools are available for managing the decompensated Type II diabetic, long term studies are needed to more precisely design their application to the individual diabetic and better elucidate the causes of treatment failure. The sequence of the application appears to be crucial.

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Benign Biliary Strictures

Nonoperative Dilatation With Balloon Catheter

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Postoperative biliary strictures in two patients were successfully dilated nonoperatively by using trans-T-tube tract approach with the Gruntzig balloon catheter. There has been no recurrence of biliary symptoms for either patient at 18 and five months respectively. It is suggested that the initial use of transhepatic or trans-T-tube tract balloon dilatation should be attempted for treating benign biliary strictures.

INTRODUCTION of the Gruntzig balloon catheter has resulted in the recognition of percutaneous transluminal angioplasty as a useful, safe procedure for treating occlusive vascular lesions of the peripheral, renal, and coronary arteries.[1-4] Because of the simplicity and safety of the procedure, the application of percutaneous dilatation has been extended to treat strictures of the bile duct, ureter, and gastrointestinal tract with promising preliminary results.[5-10] Presented here are experiences in two cases with postoperative strictures of the bile duct which were successfully treated by using trans-T-tube tract approach with the Gruntzig balloon catheter.

Case One

A 65-year-old female was referred to the Kansas University Medical Center for percutaneous removal of retained biliary stones. Six weeks prior to this admission she had undergone a common duct exploration with removal of multiple biliary stones. Two retained stones were found subsequently with a T-tube cholangiogram. In the past, she had multiple operations including cholecystectomy, abdominal hysterectomy, bilateral salpingo-oophorectomy, and internal fixation of a left hip fracture. She was diabetic and hypertensive.

Physical examination and routine laboratory studies were unremarkable except for slightly elevated blood pressure. The T-tube cholangiogram was obtained on the second hospital day and showed a stricture involving the distal common hepatic duct with a large stone proximal to the stricture *(Figure 1a)*. An additional stone was noted in the distal common bile duct. The proximal limb of the T-tube was completely dislodged from the bile duct. The stones were removed through the T-tube tract with a steerable catheter and stone basket *(Figure 1b)*.[11] After complete removal of the stones, a Gruntzig balloon catheter with a balloon 8 mm in diameter and 4 cm in length was placed at the stricture site. The balloon was inflated four consecutive times, each time for 30 seconds, up to a pressure of 5 atmospheres *(Figure 1c)*. After the dilatation, it was possible to pass a 14 F red Robinson catheter through the stricture and leave the tip of the catheter in the proximal common hepatic duct. Three days later, a repeat cholangiogram with the catheter tip placed at the proximal tube tract demonstrated a considerable dilation of the previous stricture and no residual stone *(Figure 1d)*. The catheter was placed back into the proximal common hepatic duct. The patient did well throughout the course of her hospital stay and was discharged on the seventh day. The catheter was removed one week later. The patient is 18 months post-dilatation and doing well with no recurrent symptoms.

Case Two

A 52-year-old female was readmitted to the Kansas University Medical Center for trans-T-tube tract dilatation of a biliary stricture. She had recently been discharged following a workup for chronic pancreatitis, at which time she underwent a pancreatic sphincteroplasty and T-tube placement. A T-tube cholangiogram at that time demonstrated a stricture of the proximal common hepatic duct. She continued to have intermittent epigastric pain without nausea or vomiting.

Physical examination revealed a grade I/VI systolic ejection murmur at the left lower sternal border. Diffuse tenderness was present in the mid-epigastrium with no palpable mass. A T-tube was in

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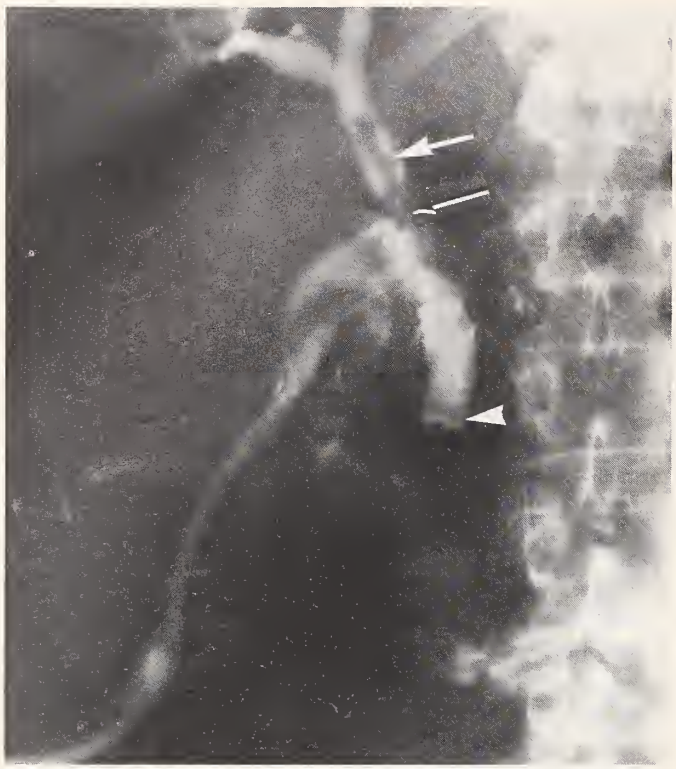


Figure 1a. The distal common hepatic duct shows a stricture (black arrow) with a large retained stone (white arrow). An additional stone is present in the distal common bile duct (white arrowhead).



Figure 1b. The stones in *Figure 1a* were completely removed.



Figure 1c. The Gruntzig balloon catheter is inflated. Notice an indentation of the balloon at the stricture site (arrowhead).



Figure 1d. Followup cholangiogram with the catheter at the proximal tube tract (small arrow) shows only a mild narrowing at the previous structure (large arrow). No residual stone is present.



Figure 2a. Initial cholangiogram demonstrates a tight stricture at the proximal common hepatic duct (arrow).



Figure 2b. One month followup post-dilatation cholangiogram demonstrates no recurrent stricture with a mild irregularity at the previous stricture site (arrow).

place in the right anterior abdomen. Laboratory studies were unremarkable. T-tube cholangiography and balloon dilatation were performed on the third hospital day. The cholangiogram demonstrated a marked stenosis of the proximal common hepatic duct *(Figure 2a)*. The hepatic ducts were slightly dilated. The T-tube was removed, and an 8 F steerable catheter was introduced into the bile duct through the tube tract. After passing a guide wire through the stricture, the steerable catheter was exchanged for a 7 F dilating catheter which was subsequently passed through the stricture. Following the initial dilatation, a Gruntzig balloon catheter with a balloon 6 mm in diameter and 4 cm in length was placed at the stricture site. The balloon was inflated three consecutive times, for 30 seconds each time, to a pressure of 5 atmospheres. The patient tolerated the procedure well. A one-day followup cholangiogram showed diffuse wall irregularity along the site of dilatation but no stenosis. The patient was discharged on the fourth hospital day with improved symptoms. The one-month followup cholangiogram showed a slight irregularity of the hepatic duct at the site of the previous stricture but no recurrent stricture *(Figure 2b)*. The drainage catheter was removed after the cholangiogram. Five months later, the patient was readmitted to the hospital with abdominal pain. She was found to have a duodenal ulcer and had no symptoms relating to the bile duct, with the total bilirubin being 0.3 mg/DL.

Discussion

Percutaneous transhepatic biliary drainage is now a well established life-saving procedure for benign or malignant obstruction of the bile duct.[12, 13] A relief of symptoms can be obtained immediately

after establishing external or internal drainage. Percutaneous drainage provides only a palliative treatment for malignant lesions. However, it can offer access for the dilatation of postoperative strictures of the bile duct or choledochoenterostomy with the balloon catheters, which may result in a cure for patients in whom surgical correction of the stricture is no longer contemplated.[5-7]

Non-operative dilatation of a biliary stricture was initially introduced by Molnar and Stockum in 1978.[5] They successfully treated nine patients with postcholedochoenterostomy strictures by using the transhepatic approach with the caged teflon catheter. Total amelioration of symptoms was achieved in eight of nine patients with no recurrence for up to five years. Teplick *et al.* and Martin *et al.* reported successful dilatation of post-choledochoenterostomy with the Gruntzig balloon catheter.[6, 7] Burhenne and Morris reported malignant biliary strictures treated with the caged catheter or Gruntzig balloon catheter. Martin *et al.* also reported percutaneous dilatation for primary sclerosing cholangitis with good preliminary results.[9]

As in the cases reported here, benign strictures following T-tube placement is a frequent complication of the surgical management of biliary and pancreatic disease. It may become a cause of successive stone formation, obstructive symptoms, and jaundice. Surgical repair of these strictures is a nuisance and is notoriously difficult with disappointing results.[14, 15] Since the T-tube tract provides an easy access to catheterization of the bile duct without significant risks or complications, initial catheter balloon dilatation should be attempted before resorting to surgical repair of the strictures. The followup in the series of Molnar and Stockum and in ours, gives considerable encouragement for the initial use of balloon catheter dilatation.

Trans-T-tube tract dilatation should be carried out four to six weeks after surgery when the tube tract is well matured with granulation tissues. This reduces the risks and complications such as perforation of the tract and peritonitis. A 14 F or larger T-tube should be placed in the right lateral position at the time of surgery. The major cause of failure in percutaneous removal of retained biliary stones or stricture dilatation is the anterior placement of a small T-tube.[11] A large, solid tube tract permits larger caliber catheters and easy manipulation of the catheter, wire, and basket. This allows more successful stricture dilatation and retrieval of co-existing retained stones, if present.

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Congenital Heart Disease

Recent Advances in Surgical Management

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SINCE ALFRED BLALOCK pioneered the era of pediatric heart surgery with the first successful systemic-pulmonary shunt for cyanotic heart disease in 1944,[1] the discipline of pediatric heart surgery has been marked by continued, frequent improvements in the ability to operate successfully on children with cardiac malformations. Outlined here are recent changes made by one author (TJB) at the University of Kansas Medical Center as well as results of the procedures on the first 41 patients with congenital heart disease who were operated upon.

Methods

Forty-one children, ranging in age from premature infancy to 14 years, were operated on for congenital heart defects at the Medical Center between April and November 1982. Sixteen of these patients had closed heart surgical procedures (those performed without cardiopulmonary bypass). These include 11 ligations of patent ductus arteriosus — nine in premature infants — and five palliative shunt procedures for cyanotic heart disease. In this group there has been one perioperative death resulting in an operative mortality rate of 6 per cent. Additionally, there was one significant surgically related postoperative complication of hemorrhage, requiring reexploration in one neonate who had undergone ligation of a patent ductus arteriosus. This hemorrhage was due to disseminated intravascular coagulation secondary to sepsis at the time of surgery.

Twenty-five of the 41 operations have been open heart procedures (those requiring cardiopulmonary bypass). Several innovative techniques, introduced at the Medical Center within the last six months, have been used in this group of patients.

There are two types of cardiopulmonary bypass technique being used. The first is standard cardiopulmonary bypass, used in patients who weigh more than 9 kg or, if less than 9 kg, have simple defects such as atrial septal defect, pulmonic valvular stenosis, or aortic valvular stenosis. Utilizing this procedure, following median sternotomy, the right atrium is doubly cannulated, and these cannulae are passed into the superior and inferior vena cavae and snared. The aorta is cannulated in a standard fashion. Cardiopulmonary bypass is begun and the patient is cooled to a body temperature of 25C. Additional topical cooling is provided throughout each procedure by iced Ringer's lactate dripped into the pericardial cradle. Once the perfusate temperature of 25C is reached, or the patient experiences ventricular fibrillation, the aorta is cross clamped in a standard fashion and iced cardioplegia is injected by hand pressure into the aortic root. The rectal temperature is about 30C at this point. The volume of iced cardioplegia utilized is 10 cc/kg on the initial injection and 5 cc/kg on subsequent injections, and these are instilled at intervals of approximately 20 minutes. Aortic root pressure is maintained at 80-90 mmHg during the cardioplegia infusions. The cardioplegia contains 30 mEq of potassium per liter and is delivered at a temperature of 5C. Following completion of the operative repair, the patient is re-

Between April and November 1982, 41 consecutive procedures were performed at UKSM-KC on children with congenital malformations. Sixteen of these procedures were performed for repair of patent ductus arteriosus or for palliation of cyanotic defects, and did not involve open heart surgery. Twenty-two procedures involved total correction (open heart) of uncomplicated malformations. There were one (6%) and two (9%) operative deaths respectively. Newly introduced techniques used to achieve these results are outlined.

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warmed and the aortic cross clamp is removed. Rectal temperature falls to 25-26C during the repair. During rewarming, when the patient's rectal temperature reaches 33.5C, cardiopulmonary bypass is discontinued, and the patient is decannulated and prepared for closure in a standard fashion. Hematocrit of the blood in the oxygenator during cardiopulmonary bypass is titrated to 25-30 per cent by the addition of citrated bank blood to standard electrolyte solution in the pump prior to perfusion. Standard cardiopulmonary bypass techniques are carried out, and the pH, PO_2, and PCO_2 are maintained within acceptable limits. Whereas in these patients aortic perfusion pressure is not as critical as it may be in older patients, we do attempt to maintain perfusion pressure during the period of bypass at 45 mmHg or greater and the cardiac index at 2.5 L/min/m^2 or greater. This technique of standard cardiopulmonary bypass is used in patients with atrial septal defect (uncomplicated), tetralogy of Fallot (when the operation is to be done through the right ventricular outflow tract), in patients with ventricular septal defect or with atrioventricular canal defect who weigh more than 9 kg, in patients with pulmonic valvular stenosis, or in patients with aortic valvular stenosis.

The second and newly introduced technique used is deep hypothermic arrest.[2] Anesthesia during this procedure is standard except for several specific points. Decadron is given intravenously (0.5 mg/kg) before bypass to help prevent cerebral edema. Lasix (0.5 mg/kg) is given intravenously at the same time. An important point is that pH and PCO_2 must be checked more frequently during deep cooling. For example, it is necessary to maintain the ventilatory rate at only 4-5 breaths per minute with the patient at 25C. The pump prime is calculated to keep the circulating hematocrit at 25 per cent. Ringer's lactate and heparinized, fresh whole blood is used. If this is not available, we use recently drawn, citrated blood. The blood volume calculation for a baby weighing 0-10 kg is 85 cc/kg. The prime volume for a baby of this weight (0-10 kg) is a total of 1,000 cc. The total dose of heparin is 3 mg/kg. Mannitol is added to the prime at a dose of 0.5 gm/kg. The flow rate during cardiopulmonary bypass (cooling and warming) is 100-150 cc/kg for a baby weighing 6-10 kg.

The technique for this procedure involves the following steps:

- Induction of anesthesia; placement of one or two central venous pressure lines and a femoral-arterial line; and placement of Foley cathether, peripheral intravenous apparatus, endotracheal tube, esophageal temperature probe, and rectal temperature probe.
- Hands, feet, genitalia, ears, and eyes are wrapped with cast felt, and the baby is covered to the sides, below and above with ice, including the head.
- The child is cooled topically to a rectal temperature of 30C or a pulse rate of 100.
- Ice is removed except from the head; the child is prepped and draped; and the chest is opened. Touching the heart is avoided unless necessary because ventricular arrhythmia is common at this temperature. Heparinization and cannulation is carried out as follows: A single atrial cannula is used. If the child weighs 5-8 kg, a 22 French is used; if the child weighs less than 5 kg, a 20 French cannula is used.
- Cardiopulmonary bypass is begun. The patient is cooled systemically and with intrapericardial topical Ringer's lactate to a temperature of 20C rectally. This will correspond to an esophageal temperature of 16-18C. Cooling must be slow to keep this small gradient.
- The aortic cross clamp is applied and 10 cc/kg of cardioplegia is injected. No additional cardioplegia is used throughout the procedure.
- The bypass pump is turned off, the aortic cannula line clamped, and the aortic infusion line is disconnected from its cannula.
- All blood is drained from the patient through the venous return cannula.
- The venous cannula is removed.
- The tubing circuit is reconnected and the perfusate is circulated at 25C bath temperature.
- Surgical repair is accomplished.
- After repair is completed, the venous cannula and the ventricular vent (if used) are replaced.
- The cardiopulmonary bypass pump is stopped, the aortic line is reconnected, and the aortic vent turned on.
- Perfusion into the aorta is started at 25C with the right atrial cannula still clamped. When the heart begins to distend, the right atrial line is unclamped and standard cardiopulmonary bypass resumed.
- The patient is warmed systemically with the perfusate temperature maintained 10 degrees greater than the rectal temperature.
- If necessary, the patient is defibrillated when the perfusate temperature is about 30C. The patient is weaned from cardiopulmonary bypass when the temperature is around 33.5C rectally. Once the bypass is stopped, the patient is decannulated and prepared for closure in a standard fashion.

TABLE I
INDICATIONS FOR DEEP HYPOTHERMIA WITH CIRCULATORY ARREST

- Weight less than 9 kg.
- Diagnosis of transposition of the great vessels, ventricular septal defect, atrioventricular canal, total anomalous pulmonary venous return, truncus arteriosus

As outlined in *Table I*, patients in whom the technique of deep hypothermic arrest has been utilized are patients weighing 9 kg or less and who have intracardiac repairs through the right atrial approach (transposition of the great vessels, complete atrioventricular canal, ventricular septal defect). Other defects that would be correctable with this technique are total anomalous pulmonary venous return and truncus arteriosus. This technique has been utilized on this service since April 1982, and there have been no known detrimental neurologic or renal sequelae in the survivors.

Results

We have performed 25 open cardiac procedures using these two bypass techniques. Of these, 22 were performed in patients with uncomplicated defects, and results of these operations are outlined in *Table II*. These patients included five with D-transposition of the great vessels, four with atrial septal defect, four with complete atrioventricular canal, three with tetralogy of Fallot, three with ventricular septal defect, one with aortic valve stenosis, one with pulmonary valve stenosis, and one with an anomalous left coronary artery. In this group of 22 patients, there were two perioperative deaths — one in a patient with transposition of the great vessels who had had a previous Blalock-Hanlon atrial septectomy, and one in a 2.8 kg patient with complete atrioventricular canal and severe pulmonary hypertension. This represents an operative mortality rate of 9 per cent. The only major postoperative surgically related complication has been the development of costochondritis in one child following repair of an atrial septal defect, and this has subsequently resolved completely. Patients with D-transposition of the great vessels were corrected using the technique of Senning.[3] Kirklin's methods were used for repair of the tetralogy of Fallot.[4] The anomalous coronary artery was repaired according to Arciniegas,[5] and McGoon's understanding of the surgical anatomy of atrioventricular canal was used in the correction of these defects.[6]

TABLE II
RESULTS OF OPEN HEART SURGERY IN 22 PATIENTS WITH UNCOMPLICATED CONGENITAL HEART DEFECTS

Diagnosis	*Number*	*Survived*	*Died*
Patients Operated on Using Standard Cardiopulmonary Bypass			
Atrial septal defect	4	4	0
Tetralogy of Fallot	3	3	0
Ventricular septal defect	2	2	0
Anomalous left coronary artery	1	1	0
Pulmonary stenosis	1	1	0
Aortic stenosis	1	1	0
Atrioventricular canal	1	1	0
Patients Operated on Using Deep Hypothermic Arrest			
Transposition of the great vessels	5	4	1
Atrioventricular canal	3	2	1
Ventricular septal defect	1	1	0
Total	22	20	2 (9.1%)

Discussion

Utilizing these two techniques of cardiopulmonary bypass and operative protection, cardiac surgery in small children with severe intracardiac defects can be carried out with a low mortality rate and few complications. Standard cardiopulmonary bypass technique is used in larger children or in those with simple intracardiac defects requiring short time periods for correction. The technique of deep hypothermic arrest is used in smaller children who have more severe cardiac defects and who require longer operative periods for correction. Deep hypothermia and complete circulatory arrest allow the surgeon to operate in a quiet, bloodless field without obstruction of vision by the atrial cannula. Additionally, in small children, the procedure greatly reduces the time required for cardiopulmonary bypass, thereby reducing the complications — such as renal, neurologic, or coagulation problems — of long bypass times. A major potential concern during deep hypothermia with circulatory arrest is the development of neurologic defects. Senning has shown that with proper cooling and limited circulatory arrest time, no major neurologic postoperative sequelae develop in survivors.[7] It appears to us that arrest times of up to 75 minutes are probably safe if core cooling is carried out carefully and completely prior to the arrest. Additionally, in these patients, the average duration of cardiopulmonary bypass was

78 minutes, and this includes the time required for cooling and rewarming. To date, using this technique, we have had no major complications that could be attributed to profound hypothermia with circulatory arrest. Also, in this group of patients there have been no episodes of postoperative hemorrhage due to coagulopathy.

Although the results presented here represent operative procedures on a relatively small number of patients, they are encouraging in that the system being developed at the University of Kansas Medical Center will likely continue to produce excellent results in pediatric cardiac surgery. The development of a pediatric cardiothoracic service requires the input and coordination of several disciplines (surgery, anesthesia, pediatric cardiology, and nursing) and the enthusiastic support of the institution. Using the techniques outlined above, long term life expectancy and productivity of patients who suffer from congenital heart disease are improved, and these children will continue to benefit from the combined expertise of the various disciplines involved in their care.

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Depression in Children

Effects of Antidepressant Therapy

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MAJOR DEPRESSIVE disorders occur in children with the same signs and symptoms as observed in depressed adults. Unfortunately there is still much unknown about this condition and its proper treatment. Consequently the National Institute of Mental Health (NIMH) has been actively promoting research in this area. An NIMH sponsored conference identified the following needs: (1) operational criteria for diagnosing childhood depression; (2) empirical testing of various treatments; and (3) assessment of age-related factors that might influence treatment response.[1] To address these needs, NIMH has sponsored individual research projects on childhood depression. Recently the University of Kansas School of Medicine was selected to conduct a placebo-control study of antidepressant treatment in hospitalized depressed children. Studies at UKSM have already shown the potential effectiveness of antidepressants in childhood depression.

Several centers have reported that operational criteria for depression similar to those for adults can be used to diagnose depression in children.[2-4] Using such criteria, as many as 30-60 per cent of prepubescent children with learning disabilities or emotional problems may be found to suffer from serious affective disorder.[4, 5] In addition to school problems and behavioral difficulties, depression may also present as vague somatic complaints without apparent organic cause. In a group of non-psychiatric, pediatric inpatients, 7 per cent were found to be depressed.[6] Thus, childhood depression apparently is not a rare disorder. The American Psychiatric Association's Diagnostic and Statistical Manual for Mental Disorders (DSM-III) third edition, includes specific criteria for the diagnosis of depression in children.

According to DSM-III, a dysphoric mood or loss of interest or pleasure in usual activities occurs in depression. A child may describe this mood as being depressed, sad, blue, hopeless, low, down in the dumps, or irritable. Depressed mood is usually prominent and persistent but not necessarily the dominant symptom. Characteristic symptoms typically include four or more of the following: poor appetite with weight loss or failure to make expected weight gain; sleep disturbance — either insomnia or hypersomnia; psychomotor agitation or retardation; apathy; loss of energy or complaints of fatigue; feelings of worthlessness, self-reproach, or guilt; diminished ability to think or concentrate; and recurrent thoughts of death or suicide. In depression, these symptoms will be present for at least two weeks. Psychiatric or medical disorders which can mimic certain features of major depressive disorder must be ruled out.

Much remains unknown about depressive disorders in children. UKSM is conducting a study of antidepressive treatment of hospitalized depressed children, and preliminary results show a potential for effectiveness of such treatment when serum drug levels are monitored and dosages appropriately regulated. Further testing is in progress.

Tricyclic antidepressants may be effective in childhood depression, but varying response rates have been reported. Ossofskey[7] reported that 49 per cent of depressed children treated with imipramine showed marked improvement. Ling *et al.*[8] reported improvement in 90 per cent of children who had depressive symptoms and headaches treated with imipramine. Weinberg *et al.*[4] reported 95 per cent of depressed children treated with tricyclic antidepressants improved while only 40 per cent of those not treated with antidepressants improved. Puig-Antich *et al.*,[9] in a more systematic study, found that imipramine in doses up to 7 mg/kg was effective in relieving depressive symptoms in 46 per cent of prepubescent children studied for five weeks.

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One factor contributing to this large variability in response rates is the lack of plasma monitoring in most studies. Such plasma drug monitoring has been studied extensively in adult depressed patients. These studies indicate a 40-fold difference may exist in the steady-state plasma drug concentration in different patients receiving the same dose of the same drug.[10] These levels correlate with the beneficial and adverse effects of the drugs. A patient with an unexpectedly low plasma level may fail to respond because he is receiving too little drug. A patient who is a slow metabolizer can become toxic on routine doses because of high drug concentration.[11] Despite advances in the psychopharmacological management of adult depressed patients, few such studies exist in children. However, plasma level monitoring of antidepressants and response to treatment has been an ongoing area of psychopharmacology research at UKSM.

It is important to study pharmacokinetics in children because one cannot extrapolate from adult data to children to determine the appropriate therapeutic plasma concentration. Children differ from adults as to several pharmacokinetic parameters: total body fat, total body water, protein binding characteristics, liver-mass to body-mass ratio, and hepatic P450 cytochrome oxidases content and activity.[12, 13] Due to their more active enzyme systems, children may produce and accumulate more metabolites of the parent drug than adult patients, and this can lead to increased cardiotoxicity.[14] Low plasma drug concentrations — due either to rapid metabolism or noncompliance — can lead to the erroneous conclusion that the patient is drug non-responsive when the concentration is too low to expect a response. As a result, an effective drug may be mistakenly deemed ineffective.

The efficacy of imipramine in treating children aged 6-12 years who fulfilled DSM-III criteria for a major depressive episode has been studied at the Medical Center. All subjects were inpatients so that medication ingestion could be closely monitored. Patients were monitored with regard to steady-state plasma levels of imipramine and its active metabolite desmethylimipramine. Such plasma monitoring permitted study of pharmacokinetic factors important in treating children with this medication. The study included three phases: (1) two weeks of inhospital observation and counseling without pharmacological treatment; (2) three weeks of treatment with a fixed dose of 75 mg of imipramine at bedtime for those who did not remit after the initial two weeks of hospitalization; and (3) three weeks of additional treatment with imipramine at an altered dosage for those who did not respond to the 75 mg dose.

Treatment during the two-week, nondrug phase consisted of: removal from the home environment by hospitalization; five one-hour individual psychotherapy sessions per week; three 90-minute group sessions per week conducted by a psychiatric nurse clinician; weekly family counselling sessions conducted by a psychiatric social worker; and occupational/recreational/milieu therapy. Throughout hospitalization, children were also instructed in a hospital-based classroom by specially trained teachers (four hours per day, five days a week). This treatment regimen was continued during the second (75 mg) and third (altered dose) phases.

Guidelines for dosage adjustment during the third phase were: decrease dose to 50 mg if no response but adverse side-effects present; or increase the dose to a maximum of 5 mg/kg if no response and no side-effects. Both three-week drug treatment phases (75 mg and altered dose) were double-blind with respect to plasma drug concentration. While all patients received active drug, neither the treating physician nor the patient knew what drug concentration was achieved by the dose given. Plasma concentrations of imipramine and its principal metabolite, desipramine, were measured using high performance liquid chromatography.

None of the 20 patients responded following the two-week nondrug treatment phase. Yet, after three weeks of treatment with imipramine, 75 mg/day, there was an 80 per cent remission rate for children whose plasma drug concentration was within a range of 125-225 ng/ml and was 0 per cent for those outside this range. At the end of the second three-week treatment period, there was a response rate of 93 per cent within this range *vs* a response rate of 25 per cent for children outside of it. Structured rating scales and clinical global impression were both used to assess response.[15, 16]

The effects of the drug on the child's blood pressure, pulse rate, and EKG were also studied. Plasma drug concentrations above 225 ng/ml routinely slowed intracardiac conduction. Prolongation of the P-R and Q-T intervals and flattening and broadening of the T-waves occurred. Concentrations above 225 ng/ml also consistently produced an increase in diastolic blood pressure and heart rate. Three patients with plasma drug concentrations above 450 ng/ml experienced potentially serious problems. One developed a mild confusional state and had EEG evidence of a drug-induced encephalopathy, and two had EKG evidence of first degree heart block.

Several important clinical guidelines are suggested by this study. First, a plasma level between

125-225 ng/ml appears desirable in the treatment of depression in children. Although response may occur below 125 ng/ml, it takes longer to occur. This lower limit is similar to the minimum effective concentration (120 ng/ml) reported for adults.[15] Second, levels above 225 ng/ml appear to cause undesirable side-effects without further therapeutic benefit.

The NIMH sponsored study currently underway at the UKSM Medical Center will test the effectiveness of imipramine *vs* placebo in the treatment of depression in children. As in the earlier study, subjects will be depressed children aged 6-12 years who are in good physical health and willing to participate. They will be randomly assigned to treatment with either imipramine or placebo. The laboratory will adjust the dosage so that blood level will be in the 125-225 ng/ml range for those on imipramine. However, the treating physician will be "blind" as to whether the patient is receiving imipramine or placebo. Subjects will be closely monitored for side-effects and for improvement or worsening of depression. Acute treatment will proceed for six weeks. For those who respond, an outpatient follow-up phase is planned to determine the relapse risk once the medication has been discontinued. This study should provide important information on imipramine treatment of depressed children.

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Ureteral Calculus Extraction

A Non-Surgical Percutaneous Approach

HILTON I. PRICE, M.D. *and* **KYO RAK LEE, M.D.,** *Kansas City, Kansas*

PERCUTANEOUS NEPHROSTOMY is a well established technique in the management of obstructive uropathy.[1, 2] The nephrostomy tract offers not only a means of urinary diversion but also a direct access to the renal collecting system.[3]

Percutaneous removal of urinary calculi via a nephrostomy tract offers a safe alternative to surgery. We present an 86-year-old woman with multiple medical problems in whom urate calculi were successfully removed percutaneously.

Percutaneous urinary calculus extraction is a safe and useful technique in patients who are poor surgical risks and in patients with retained renal calculi following nephrolithotomy.[3-5] In the latter group of patients, re-operation is often technically more difficult than the primary procedure due to scarring and fibrosis in and around the kidney.[4, 6, 7]

We present a patient with obstructive uropathy due to urate calculi with resultant renal failure in whom percutaneous ureteral calculus extraction was successfully performed effecting improvement in the patient's renal function.

Case Report

An 86-year-old female was transferred to the Kansas University Medical Center from an outside hospital with a diagnosis of acute renal failure and septicemia. Klebsiella was cultured from both urine and blood, and the patient was placed on appropriate antibiotics. She was in mild congestive cardiac failure with evidence of atrial fibrillation.

Biochemical evaluation revealed BUN, 215 mg/dl (normal 8-20); serum creatinine, 16.2 mg/dl (normal 0.5-1.2); and blood uric acid level of 15.6 mg/dl (normal 2.0-6.0).

A diagnosis of obstructive uropathy due to urate



Figure 1a. Antegrade pyelogram through a left sided nephrostomy tube demonstrates dilatation of the calices and ureter with numerous radiolucent calculi in the distal ureter (arrows). The contrast material is not seen entering the bladder.

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calculi was suspected, and a retrograde pyelogram was performed. A partial obstruction of the right proximal ureter and a complete obstruction of the left ureter at the pelvic brim was demonstrated. An attempt at basket stone extraction was unsuccessful.

A left percutaneous nephrostomy was performed, and the urine was noted to be cloudy and infected. An antegrade pyelogram confirmed the presence of radiolucent calculi in the distal left ureter.

The patient was treated with allopurinol and urinary alkalinizing agents. A repeat nephrostogram three weeks later demonstrated little significant interval change in the ureteral calculi *(Figure 1a)*. The patient was assessed to be a poor surgical risk, and a percutaneous stone extraction was performed.

Using the nephrostomy tube as the site of entry into the renal collecting system, with a combination of guidewires and catheters, a Dormia stone basket was positioned distal to the ureteral calculi, in the urinary bladder. The stone basket was opened and then carefully withdrawn up the left ureter, bringing a partly fragmented calculus with it. Two further passages of the stone basket retrieved the remaining calculi. The pelvi-ureteric system was then flushed with saline to remove any remaining debris. An 8.3 French pigtail nephrostomy catheter with multiple side holes (Cook) was left in the left renal pelvis.

A repeat antegrade nephrostogram three days later revealed no residual calculi, and there was free drainage of contrast medium into the urinary bladder *(Figure 1b)*. The nephrostomy tube was removed, and the patient was later discharged from the hospital with significant improvement in renal function.

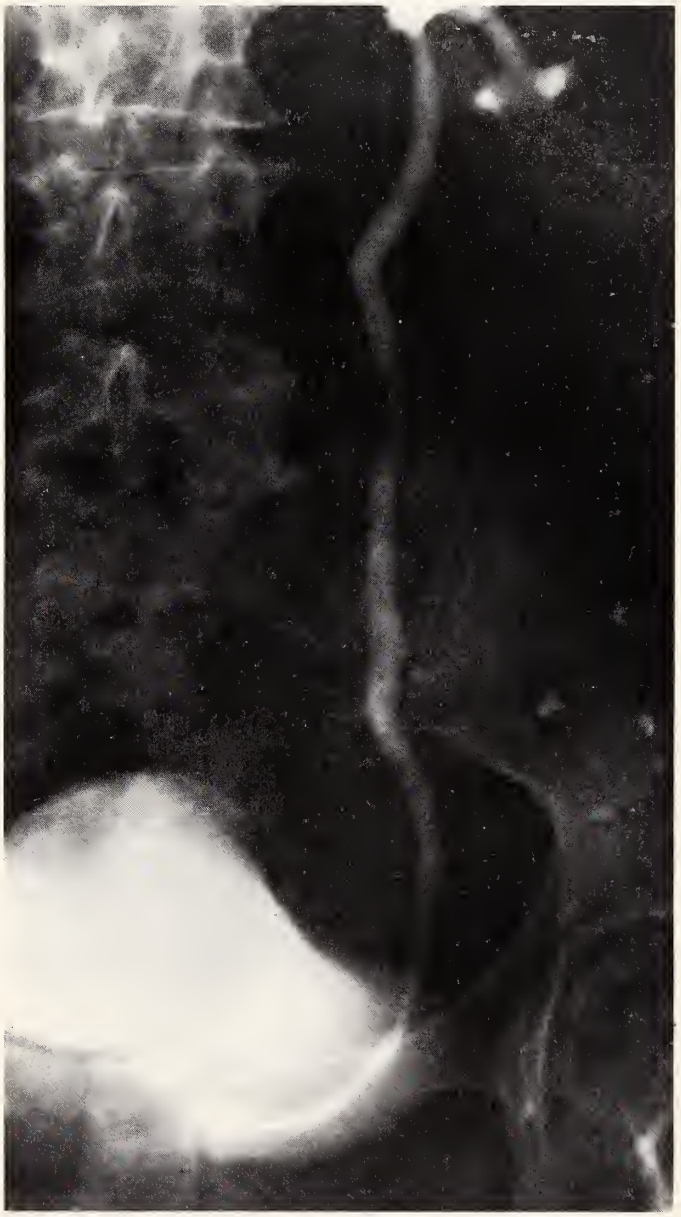


Figure 1b. Three day followup pyelogram demonstrates no residual calculi with free passage of the contrast material into the bladder. The collecting system has returned to normal.

Discussion

The non-operative percutaneous management of pyeloureteral stones depends on the chemical composition, size, and site of the calculus.[5] Uric acid, cystine, and struvite stones may be dissolved by irrigation with specific solvents administered through the percutaneous nephrostomy catheter.[2, 3, 5] This requires prolonged hospitalization, is not without risk, and is not always successful.[6]

Insoluble calculi may be either extracted or displaced from the renal pelvis or ureter.[2, 3] These non-operative techniques are especially useful in patients who are poor surgical risks, patients with retained stones following pyelolithotomy, and patients with recurrent calculus formation.[4, 5]

Percutaneous stone extraction can be performed using sedation and local anesthesia on an outpatient basis and has few significant complications.[2] These include transient hematuria and fever and, rarely, ureteral damage.[5] These complications can be minimized by using prophylactic antibiotics and a gentle technique.[2]

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(Continued on page 146)

Colorectal Carcinoma

The Role of Radiation Therapy

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Colon and rectal carcinoma is one of the most common internal malignancies in the United States. The American Cancer Society estimates that 85,000 new cases were diagnosed in 1982 in the United States resulting in approximately 49,000 deaths.[1] It is estimated that it will occur in approximately 5 per cent of U.S. born males and 6 per cent of females.

Miles devised an abdominoperineal resection for the treatment of rectosigmoid colon cancer in 1908. Since then the choice of primary curative treatment has been surgery. Unfortunately, there has been no significant improvement in the overall cure rate in the last 30-40 years by surgery alone, despite significant advances in surgical techniques. However, knowledge of the natural history of colorectal cancer has increased during this time. Usually, local recurrences are the main problem, with 25-50 per cent of surgical failure resulting in death from local recurrences alone. At present, there is nothing more that can be offered to prevent pelvic recurrences by conventional surgical methods alone.[2]

Staging

Cuthbert E. Dukes[3] in 1932, after a meticulous gross and microscopic study of several hundred cases at St. Marks Hospital, London, described a staging classification based on depth of invasion and involvement of lymph nodes that consisted of three stages: *i.e.* stage A — the tumor involving mucosa and submucosa; stage B — invasion into muscularis propria (circular and longitudinal muscle); and stage C — with involvement of regional lymph nodes. This staging system is entirely surgical and pathological. In 1954 Astler-Coller[4] modified the Dukes classification which was then further modified by Gunderson and Sosin in 1974 as shown in *Table I*.

Treatment

Surgery is the modality of choice in a curative treatment for cancer of the colon and rectum. In early lesions, three of four patients can anticipate a five-year disease-free survival.[6] Unfortunately, a majority of patients present with advanced disease with tumor that has spread beyond intestinal walls to adjacent lymph nodes, contiguous organs, or to remote areas by the time the diagnosis is made *(Table II)*. In these patients, the surgical outlook is very poor, and effective adjuvant therapy should prove useful.

Surgery is the modality of choice in curative treatment for cancer of the colon and rectum. Since a majority of patients present with advanced disease with a poor surgical outlook, adjuvant therapy may be warranted. Clinical and experimental data demonstrate the benefits of preoperative radiation therapy and some clinical reports indicate the beneficial effects of postoperative radiotherapy.

Radiation Therapy

Symmonds[7] in 1914 used radium to treat adenocarcinoma of the rectum. Later, in 1928, Neumann and Coryn[8] reported a number of patients whose inoperable growths were changed into the operative category by means of preliminary treatments with radium. Adenocarcinomas of the colon and rectum are radioresponsive tumors, although they respond slowly, thereby creating an impression of radioresistance. Although the tumor tissue and the surrounding normal tissues are approximately equal in their sensitivity to ionizing radiation, the normal mucosa has a large capacity to repair sublethal damage during fractionated x-ray therapy.[9] Numerous investigators have explored the use of radiation therapy in the treatment of carcinoma of the rectum, either as an adjuvant to surgical resection,[10-13] for palliation,[14-16] or in some cases as a curative treatment.[17-22] Radiation therapy has been chosen in the following forms: as a primary curative treatment; as an adjuvant therapy either as a preoperative or postoperative course; and as a palliative treatment.

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TABLE I
STAGING: CARCINOMA OF THE COLON AND RECTUM

Dukes (1932)	*Astler-Coller (1954)*	*Gunderson & Sosin (1974)*
A—Invasion of mucosa and submucosa	A—Same	A—Same
B—Invasion of Propria	B1—Invastion of muscularis propria	B1—Same
	B2—Invasion through entire thickness of bowel wall	B2—Same
C—As in A & B but with positive nodes	C1—As B1 with positive nodes	B3—Tumor outside the bowel wall
	C2—As B2 but with positive nodes	C1—As in C2 B but with C3 positive nodes
	D—Distant Metastases	D—Distant

Curative

Surgery is generally accepted as the curative treatment in carcinoma of the colon and rectum regardless of the size of the primary tumor. However, there is limited information regarding definitive curative treatment of rectal carcinoma by radiation therapy alone. Binkley[17] reported a 50 per cent five-year cure rate in 34 evaluable patients. Williams[20] described his experience with megavoltage therapy in treating 189 patients with a majority of the patients in advanced stage with tumor extension beyond the rectum to neighboring structures or involving lymph nodes, and/or with distant metastasis. Symptomatic relief of pain, bleeding, or tenderness was obtained in 70-80 per cent of cases and colostomy was avoided in about 40 per cent of cases. Recently, Wang[19] cited seven patients who survived from 4-15 years without disease. The patients were treated for inoperable, recurrent, or residual disease. Using the special contact x-ray tube, Papillon,[18] from 1951 to 1972, treated 186 patients with tumors less than 5 cm in size, well differentiated, confined to the wall of the gut, and located in the distal 12 cm of the rectum. Of the 133 patients followed for more than five years, 104 (78%) were alive, well, and disease free with normal intestinal function. Only three of these survivors received surgical treatment for local failure.

TABLE II
CARCINOMA OF THE RECTUM AND RECTOSIGMOID COLON: STAGING AND SURVIVAL[21]

Stage	*Incidence %*	*Survival %*
A	25-35	70-90
B	25-35	35-45
C	35-45	20-30

A combination of external and contact radiation treatments has been used in selected patients with a carcinoma of the rectum.[21] The advantages include elimination of the necessity for a permanent colostomy. In case of failure, electrocoagulation and abdominoperineal resections are still available alternatives. On the basis of present information, it seems reasonable that patients who are poor surgical risks, or have inoperable tumors, or in a selected group as described above, be considered for radical radiation therapy.

Preoperative Irradiation

The rationale for preoperative radiation therapy includes: an increase in resectability; prevention of seeding during surgery; and destruction of microscopic foci of tumor that are beyond surgical margins or in the lymph nodes. All have the goal of decreasing local recurrences and therefore increasing survival.

In addition to these theoretical considerations, clinical and experimental evidence suggest benefits from preoperative radiation therapy. In 1964, Powers and Tolmach[22] reported on an animal study and suggested that a single preoperative dose of 500 rads improved survival time. Agostino and Nickson[23] showed a decreased local recurrence rate and improvement in survival in an animal colon tumor model. Similar observations were recorded by Hoye and Smith[24] and Inch and McCredie.[25] Beneficial effects of preoperative radiation therapy in rectosigmoid carcinomas were demonstrated in an early retrospective, nonrandomized study by Stearns *et al.*[26] It showed a better survival rate for Dukes stage C patients in a preoperative radiation therapy group when compared to surgery alone (37% vs 23%). This study encouraged others to consider radiation therapy as part of the management of rectal cancer *(Table III)*. A subsequent randomized study by Stearns *et al.*[27] did not show improvement in survival. This study failed to take advantage of proper

TABLE III
CARCINOMA OF THE RECTUM AND RECTOSIGMOID COLON: FIVE-YEAR SURVIVAL SURGERY *VS* PREOPERATIVE RADIATION THERAPY (RT) AND SURGERY (LITERATURE REVIEW)

Study	*Stage*	*Surgery (%)*	*Preoperative and Surgery (%)*	*RT dose (rads/week)*
Stearns *et al.* (1959)	A and B	62	68	1,500/2 wks
	C	23	37	
Stearns *et al.* (1968)	A and B	70	63	2,000/2 wks
	C	41	34	
Roswit *et al.* (1975)	Total resections	32	40	2,000-2,500/
	Curative resections	39	48	2-2.5 wks
Antunez *et al.* (1973)	A and B	-	87	800 x 3/3 wks
	C	-	23	
Stevens *et al.* (1976)	Curative resections	38	53	5,000-6,000/ 5-6 weeks
Kligerman *et al.* (1972)		30	41	4,500/4.5-5 weeks
Ruff *et al.* (1961)			44	Radium alone
Rider *et al.* (1977)	All	35-40	35-40	500 rads single
	C	20	40	dose
Dedkov and Zibina (1976)	All (3 yrs)	60	79	3,000 (500 x 6)
	C (3 yrs)	37	77	over 1.5 wks

randomization and stratification methodology and therefore remains difficult to interpret because of bias.[28] Also, for the purposes of statistical analysis, the 347 randomized and 443 nonrandomized patients have been mixed. Although no reported series can claim a difference that is highly significant, the presence of improved results in most series warrants a serious consideration of preoperative radiation therapy as an important therapeutic modality. The main disadvantage of this type of therapy is that, when preoperative irradiation is given to all patients with the diagnosis of carcinoma of the rectosigmoid, about 50 per cent of the patients with early stages may be unjustifiably subjected to this treatment.[29, 30]

Postoperative Radiation Therapy

The rationale of postoperative radiation therapy is that: the extent of disease is defined and radiotherapy may be spared for very favorable early cases or for those patients with widely disseminated disease; the area of gross residual disease can be defined by surgical clips to help in radiation therapy planning in order to spare normal structures such as the small bowel; and the extent of disease is not altered so that the surgeon is not biased in favor of lesser, possibly inadequate, surgery.

The possible disadvantages of postoperative radiation therapy are: tumor cells may be in an hypoxic state after surgery and thus would be less radiosensitive; more complications may arise as a result of small bowel fixation after surgery within the radiation field; and the advantages of preoperative radiation therapy would be lost.

Early studies *(Table IV)*[31-34] demonstrate a low recurrence rate in patients at high risk for failure when postoperative irradiation was given in the range of 4,500-6,000 rads. Ghossein *et al.*[33] reported on 125 patients with locally advanced disease (B-2, C-1, and C-2) of which 78 patients had rectosigmoid carcinoma and 47 had proximal colon cancer (above the mid sigmoid colon). For patients with rectosigmoid carcinoma, the pelvis and para-aortic areas were treated with a dose of 4,600 rads in 4.5-5.0 weeks, whereas total abdominal irradiation was used for patients with proximal colon cancer. The average followup was 38 months for patients with no residual disease and with microscopic disease; local control was excellent — in the range of 77-84 per cent. But for patients with gross residual disease, the local control rate was only 50 per cent. The overall control rate was 64 per cent (80/125). The preliminary results of a gastrointestinal study group (GITSG71-75)[35] shows statistically signifi-

TABLE IV
CARCINOMA OF THE RECTUM AND RECTOSIGMOID COLON, LOCAL CONTROL WITH POSTOPERATIVE RADIOTHERAPY (LITERATURE REVIEW)

Study	*Stage**	*Patients (Number)*	*Local Control (%)*	*Followup (months)*
Mendiondo *et al.*[31] (1976)	B2 and B3	15	60 (9/15)	9-54
	C2 and C3	12	33 (4/12)	4-18
Turner *et al.*[32] (1977)	B2, C1, and C2	40	60 (25/40)	16-25 (Average)
Ghossein *et al.*[33] (1981)	B2, C1, and C2		64 (80/125)	38 mo. average followup
Withers and Romsdahl[34] (1981)	3A	20	100 (29/29)	1-8 years
	3B	44	85	(followup period)

cant prolongation of disease free survival. Patients receiving both irradiation and chemotherapy continue to have fewer systemic and local recurrences compared to those treated with surgery alone.

"Sandwich" Radiation Therapy

Even a single low dose of 500 rads as preoperative therapy by Rider *et al.*[36] showed an improvement in survival, especially for stage C patients. Based on these results, Brenner *et al.*[37] and Mohiuddin *et al.*[38] attempted a sandwich technique which consists of low dose preoperative radiation therapy, curative surgery, then postoperative radiation therapy where indicated. Earlier results as reported by Brenner *et al.*[37] using 2,000 rads preoperatively with an additional 2,600 rads following surgery for Dukes B and C lesions, showed no unusual complications, and there were no local recurrences in 26 patients who received a complete course. Mohiuddin *et al.*[38] used 500 rads preoperatively in a single dose followed by surgery in one to three days, then postoperative irradiation to a dose of 4,500 rads in conventional fractionation for stages B-2, C-1, and C-2. None of the 13 patients who received a complete course developed any pelvic recurrences. These approaches clearly merit randomized clinical trials.

Palliative Radiation Therapy

Pelvic recurrence after surgical excision of cancer of the rectum or rectosigmoid occurs most frequently within the first two years after surgery. Its occurrence generally indicates an extremely poor prognosis. Among the most common symptoms and signs of reactivation of the disease are pain, recurrent mass, bleeding, tenesmus, and mucous discharge. In such situations, radiation therapy can give maximum palliation with minimum discomfort. This is particularly true in patients with disease localized in the pelvis. With respect to pain, more than 80 per cent of patients respond favorably to a modest radiation dose (3,000 rads in three weeks). Bleeding is controllable with a similar dose of radiation in 80-90 per cent of cases; mass lesions required at least 4,000 rads in four weeks. About 50 per cent of patients with symptomatic liver metastases benefit from palliative radiotherapy. Urdaneta-LaFee *et al.*[15] analyzed the results of palliative radiation therapy in 102 cases of primary inoperable rectal and rectosigmoid cancer and recurrent carcinoma of the rectum. Symptomatic relief followed irradiation in 80 per cent of patients; overall survival was 5 per cent; and colostomy was avoided in 71 per cent of cases.

Summary

Surgery is the mainstay of treatment in colorectal cancer. In high risk patients with locally advanced disease — stage B-2 and C — adjuvant postoperative radiation therapy seems to minimize the local failure rate, thus improving ultimate survival. Preoperative radiation therapy is indicated in obviously unresectable rectal cancers. Radiation therapy offers an excellent palliation of pain, bleeding, and other discomforts.

References are available from the authors.

Breast Cancer Screening

Evaluation of Kansas Program Data

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The Breast Cancer Detection Center was established at the University of Kansas Medical Center as part of the Breast Cancer Detection Demonstration Project (BCDDP), a national multicenter program funded jointly by the National Cancer Institute (NCI) and the American Cancer Society (ACS). The purpose of the project was to disseminate techniques of early detection of breast cancer both to the public and to the medical profession. The University of Kansas BCDDP was the tenth of 29 centers (27 locations) to be funded throughout the United States. Each center was responsible for recruiting approximately 10,000 women during a two year period with a mandate to screen each woman for breast cancer on an annual basis for five years. Participants were screened using a combination of medical history, physical examination, lo-dose film mammography, and thermography to detect breast cancer in its earliest stages. Breast self examination (BSE) instruction was also provided at the screenings, and participants were encouraged to practice BSE at home on a monthly basis.

During the five year screening period, the Kansas Breast Cancer Detection Center performed 38,611 annual screening examinations on 10,376 women. Of the 105 counties in Kansas, 60 were represented by screenees in the Kansas BCDDP. There were also more than 1,600 participants from Kansas City, Missouri, 700 from other Missouri towns, and more than 100 women from other states. At the fifth year of the screening period, 5,835 (56.2%) were still actively participating in the program.

A demographic profile of the screenees *(Tables I* and *II)* reveals that although every woman who wanted to be screened was accepted into the Detection Center, most participants were between the ages of 35 and 74 years at entry. The median age of the participants was 50.3 years. The great majority of the Kansas BCDDP screenees were white (92.3%); only 3.2 and 0.2 per cent of the population were black and oriental, respectively. The remainder of the women were categorized as "other race" or "race unknown." Most of the women were married (82.0%) at the time of entry into the program. More than one-half of the screenees (52.0%) had attended high school, and 40.7 per cent had attended college for one or more years. Data on household income levels indicated that 47.4 per cent of the women were from households with incomes less than $15,000/year, while 43.3 per cent of them had incomes greater than $15,000/year.

The University of Kansas Breast Cancer Detection Demonstration Project was one of 29 such centers funded jointly by the National Cancer Institute and the American Cancer Society. The purpose of the project was to disseminate techniques of early detection of breast cancer both to the public and the medical profession. The screening program spanned a five year period.

As part of the screening process, participants were requested to complete a patient history form during their first visit and then update the form during each subsequent visit. The initial patient history record contained questions regarding relevant medical history prior to entry into the program. A 10 per cent sample of these records (N = 949) was analyzed and a summary of the data is presented in *Table III*. A majority of the screenees indicated they had received at least one breast examination by a physician prior to entering the program (89.5%), although only a small minority (16.3%) had ever received a mammogram. Even fewer (9.2%) had received more than one mammogram.

Although the program was originally designed to screen asymptomatic women, the Kansas BCDDP did not deny access to women who were concerned about potential signs or symptoms. A small percentage (5.8%) of women sampled recorded an aware-

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TABLE I
DEMOGRAPHIC PROFILE OF KANSAS BCDDP PARTICIPANTS*

Age	*Number of Annual Screenings*	*Per Cent*
<35	220	0.6
35-39	4,453	11.5
40-44	5,583	14.5
45-49	6,299	16.3
50-54	6,427	16.6
55-59	4,985	12.9
60-64	3,602	9.3
65-69	2,499	6.5
70-74	1,276	3.3
75-79	170	0.4
>79	53	0.1
Unknown	3,044	7.9
TOTAL	38,611	100.0
Race		
White	25,634	92.3
Black	1,252	3.2
Oriental	85	0.2
Other/NA	1,640	4.2
TOTAL	38,611	100.0

* Data on age and race are taken from final screening recommendation forms and differ slightly from data on education, household income, and marital status which are taken from initial patient history records.

ness of a breast lump at entry into the program, and 17.2 per cent reported undergoing previous breast surgery. Of the 10 per cent sample, 7.5 per cent indicated having some form of cancer prior to entry and 2.0 per cent of the sample indicated having previous breast cancer. A question regarding family history of breast cancer was also included on the initial patient history record, and 25.6 per cent of the participants reported one or more relatives with a history of breast cancer. "Family history" is defined as breast cancer occurring in grandmothers, mothers, sisters, half-sisters, daughters or aunts; however the initial patient history record did not always indicate the specific relationship.

The instruction of BSE was an important component of the Kansas BCDDP, and women were asked on the initial patient history record whether they practiced BSE prior to joining the program. Of the women sampled, 31.9 per cent reported practicing BSE on a regular basis, and only 16.4 per cent reported never doing BSE. These figures are similar to a National Institutes of Health survey which reported that 29 per cent of women practiced BSE on a monthly basis, and 23 per cent had not practiced BSE during the past year.[1]

TABLE II
DEMOGRAPHIC PROFILE OF KANSAS BCDDP PARTICIPANTS*

	Number of Participants	*Per Cent*
Household Income		
<$5,000	810	8.1
$5,000-$10,000	1,569	15.6
$10,000-$15,000	2,379	23.7
$15,000-$30,000	3,249	32.4
$30,000-$100,000	1,060	10.6
>$100,000	35	0.3
Uncertain/no response	940	9.4
Total	10,042	100.0
Education		
1-8 years	600	6.0
9-12 years	5,226	52.0
1-3 years college	2,337	23.3
4 years college	1,068	10.6
5+ years college	678	6.8
Uncertain/no response	133	1.3
Total	10,042	100.0
Marital Status		
Married	8,232	82.0
Single	291	2.9
Divorced	445	4.4
Separated	73	0.7
Widowed	996	9.9
No response	5	0.0
Total	10,042	100.0

* Data on age and race are taken from final screening recommendation forms and differ slightly from data on education, household income, and marital status which are taken from initial patient history records.

Surgical Recommendations and Performance

When a woman's screening examination resulted in one or more modalities showing suspicious findings warranting further investigation, the Kansas BCDDP made a recommendation for surgery. The recommendation, however, was a nonspecific recommendation and did not always specify a particular procedure (*i.e.*, biopsy, aspiration, or surgical consultation). The surgical recommendation rates by age at entry and year are presented in *Table IV*. In year one, the number of surgeries recommended per 1,000 annual screenings is much greater for women over 50 years of age at entry than for women under 50 years of age. This may reflect the higher prevalence of cancer in older women. For years two through five, however, the rates are fairly comparable, and there is a general downward trend each year, suggesting that the Kansas BCDDP became

TABLE III
RELEVANT MEDICAL HISTORY OF KANSAS BCDDP PARTICIPANTS PRIOR TO SCREENING*

Medical History	*Response*	*Per Cent*
Number of breast examinations by a physician	0	4.2
	1	2.6
	2	5.2
	3 or more	81.7
	Uncertain/no response	6.3
Number of mammograms	0	83.2
	1	8.1
	2	2.6
	3 or more	5.6
	Uncertain/no response	0.3
Breast mass at entry	No	87.4
	Yes	5.8
	Uncertain/no response	6.8
Number of breast surgeries	0	82.4
	1	12.6
	2 or more	4.6
	Uncertain/no response	0.3
Previous history of breast cancer	0	96.7
	1	1.8
	2 or more	0.2
	Uncertain/no response	1.3
Previous history of cancer†	None	91.4
	Once or more	7.5
	Uncertain/no response	1.2
Number of relatives with history of breast cancer (includes grandmothers, mothers sisters, half-sisters, daughters or aunts)	0	72.6
	1	17.5
	2	3.1
	3 or more	5.0
	Uncertain/no response	1.9
Breast self-examination	No	16.4
	Yes, few times	49.0
	Yes, regularly	31.9
	Uncertain/no response	2.6

* These figures are based on a 10 per cent sample of initial patient history records.
† Includes breast cancer.

more proficient at screening and more discriminating when making a surgical recommendation for a biopsy, aspiration, or surgical consultation. When surgery was recommended for a woman in the Kansas BCDDP, three of four women carried through and actually had a surgical procedure performed (either a biopsy or aspiration).

Biopsies used in the analysis were performed as a result of a surgical recommendation by the project at an annual or early recall screening, or when a woman saw a surgeon prior to a scheduled early

TABLE IV
SURGICAL RECOMMENDATION RATES FOR WOMEN <50 and ≥50*

	Surgeries Recommended			
	Age at Entry <50		*Age at Entry ≥50*	
Year	*Number*	*Rate†*	*Number*	*Rate†*
1	114	23.2	215	43.7
2	56	15.5	72	17.6
3	58	18.0	55	14.8
4	26	9.1	29	8.6
5	12	4.9	23	7.9

* These rates are not directly comparable to the cancer detection rates and biopsy rates, since they include recommendations for biopsy, aspiration or surgical consultation, and they do not include recommendations for early recall examination.
† Rates are per 1,000 annual screenings.

recall screening to follow up an abnormal modality finding. Early recall is defined as a scheduled followup mammogram and/or physical examination within six months of an annual screening examination at which an abnormal modality finding was noted. The biopsy rates are not directly comparable to the surgical recommendation rates because the latter include recommendations for aspirations and surgical consultations, and do not include recommendations for early recall examinations.

Cancer Detection

There were a total of 134 cancers found in the Kansas BCDDP population. Of these, 111 were detected as a result of the screening program and 23 were uncovered outside the program. Cancers are defined as "Project Detected" if they were detected as a result of compliance with a surgical recommendation made during an annual screening or early recall examination, or if the cancer was detected when a woman who was scheduled for an early recall examination due to an abnormal modality finding chose to see a surgeon prior to the scheduled examination.[2] Cancers diagnosed outside the program are separated into two categories: (1) interval cancers which include those diagnosed within one year after an annual screening at which no surgical recommendation was made (N = 20); and (2) post screening cancers which include those diagnosed more than one year after a woman's last annual screening (N = 3).

All the cancers have also been stratified by lesion size and categorized into four groups: (1) noninfiltrating cancers; (2) infiltrating cancers less than 1 cm

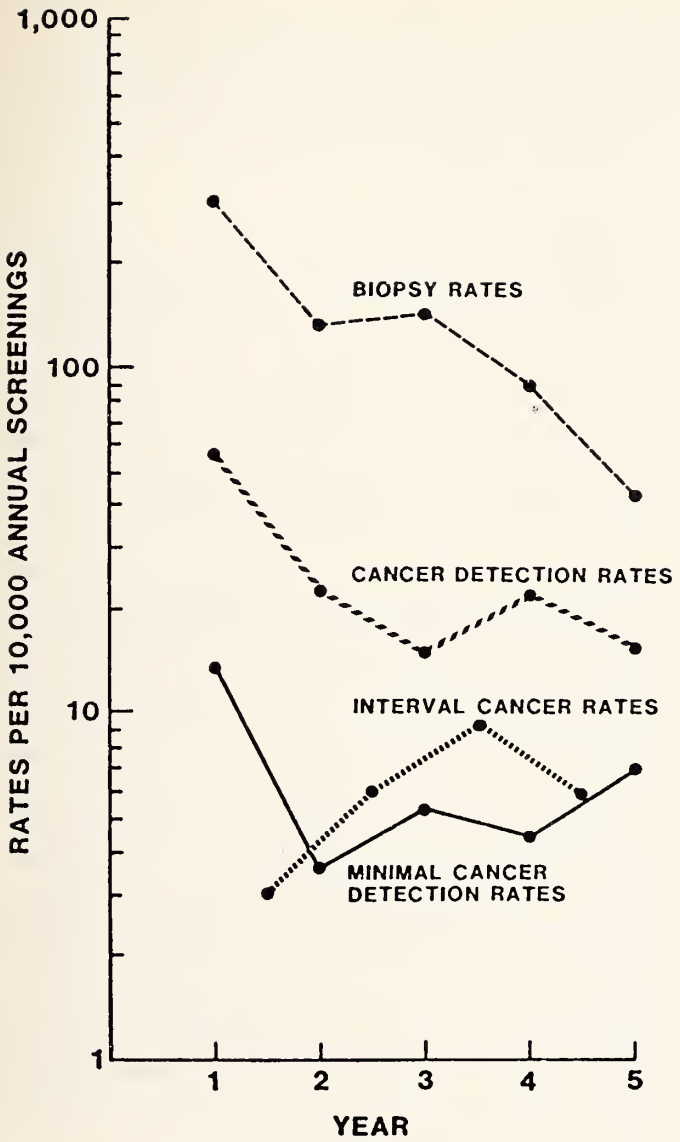


Figure 1. Comparison of crude cancer detection rates, biopsy rates, minimal cancer detection rates, and interval cancer rates for years one to five.

in diameter; (3) infiltrating cancers greater than 1 cm in diameter; and (4) size unspecified cancers. For parts of the analysis, cancers that were noninfiltrating or infiltrating less than 1 cm will be referred to as "minimal cancers." These cancers account for 25.2 per cent of the cancers detected by the Kansas BCDDP.

Figure 1 and *Table V* compare the biopsy rates, cancer detection rates, minimal cancer detection rates, and interval cancer rates for years one through five of the Kansas BCDDP program. The biopsy, cancer detection, and minimal cancer detection rates are much higher in year one than in years two through five and reflect the difference between prevalent and incident cases. The biopsy rates decrease disproportionately in years four and five suggesting the Kansas BCDDP team became more effective at detecting breast cancer. This is also evidenced by the increasing rate of minimal cancers detected in the latter years of the program. The interval cancer rates are depicted at mid-year since these cancers were discovered between annual screenings. Although the rates appear to rise slightly each year, only 20 interval cancers were found throughout the program period and the consequent small numbers of interval cancers in the numerators produce unstable rates and make interpretation difficult.

Figure 2 and *Table VI* present the age-specific breast cancer detection rates for years one through five combined. (Cancers detected in the "age unknown" category have been excluded from this graph.) The breast cancer detection rates generally increase with age as has been noted by many previous authors.[3] At approximately ages 50-54 years, however, the rate decreases and then begins to increase again. This phenomenon was first noted by Johannes Clemmesen in 1948, and has become

TABLE V
COMPARISON OF CANCER DETECTION RATES, BIOPSY RATES, MINIMAL CANCER DETECTION RATES AND INTERVAL CANCER RATES FOR YEARS ONE TO FIVE

*Rates**	*Year* 1	1.5	2	2.5	3	3.5	4	4.5	5
Biopsy rates	297.0		133.7		144.4		88.8		42.8
Cancer detection rates	56.1		22.9		14.6		22.2		15.4
Minimal cancer detection rates†	13.8		3.6		5.3		4.4		6.9
Interval cancer rates‡		3.0		6.0		9.3		5.9	

* Rates are per 10,000 annual screenings.
† Minimal cancers are defined as noninfiltrating cancers or infiltrating cancers less than 1 cm in diameter.
‡ Includes those cancers discovered outside the Project within one year after an annual screening.

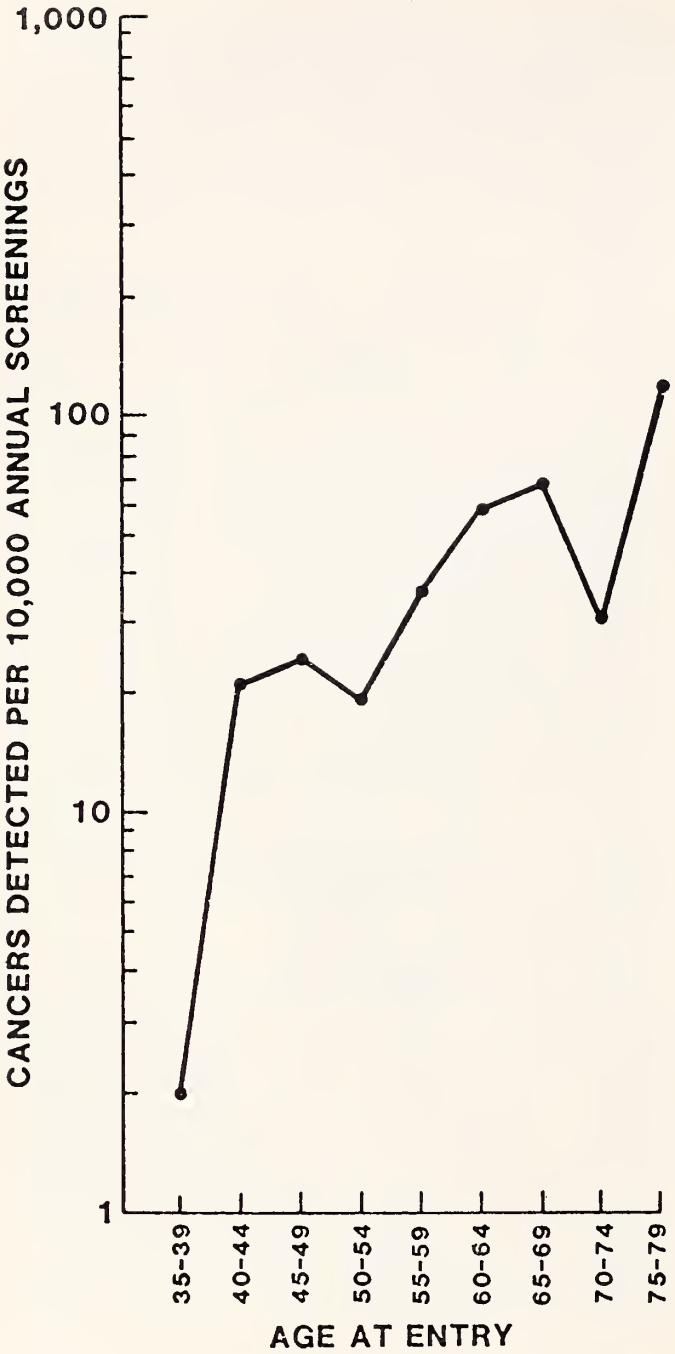


Figure 2. Age-specific breast cancer detection rates.

known as "Clemmesen's Hook."[4] It has been seen in some data sets since then but not in others.[3] Although there is no conclusive evidence for the cause of this phenomenon, Clemmesen believed that hormonal changes occurring at menopause account for the decrease in the rate.[4] Other researchers claim the "hook" suggests the possibility of two types or components of breast cancer, one premenopausal and one postmenopausal.[5, 6] Still others purport "Clemmesen's Hook" as being part of a cohort phenomenon.[7, 8] The decrease that occurs in the Kansas BCDDP data in the 70-74 year age group is probably an artifact due to the low numbers of cancers detected in that age group (4).

Detection Methods

When the Kansas BCDDP opened in 1973, a typical screening examination consisted of a medical history, physical examination, lo-dose film mammography, and thermography. Two changes occurred in 1977 which affected that routine. First, thermography was eliminated as a routine screening modality, and restrictions were placed on the use of mammography for women under 50 years of age at the time of the examination. Because of a perceived risk of radiation exposure and because no reduction in mortality was found in women under 50 during the Health Insurance Plan of Greater New York study conducted in the 1960s,[9] it was decided that the benefits of mammography did not outweigh the risks for younger women. For women under age 50 years, only those who were at high risk for breast cancer were eligible for routine screening with mammography after 1977. Women younger than 50 years of age were considered "high risk" if they met the following criteria: (1) Women ages 35-39 were at high risk if they had a personal history of breast cancer, or if they had an abnormal physical examination; (2) Women ages 40-49 were considered at high risk if they had an abnormal physical finding, had a personal history of breast cancer, or had a mother or sister who had a history of breast cancer.

Women older than 50 years of age were still eligible for routine mammography, but the concern about radiation exposure decreased the number of eligible

TABLE VI
AGE-SPECIFIC CANCER DETECTION RATES

Age at Entry	*Breast Cancer Detected Per 1,000 Annual Screenings*	
	Number	*Rate*
35-39	1	0.2
40-44	12	2.1
45-49	15	2.4
50-54	12	1.9
55-59	18	3.6
60-64	21	5.8
65-69	17	6.8
70-74	4	3.1
75-79	2	11.8
Age Unknown	9	3.0

TABLE VII
BREAST CANCERS DETECTED BY THE KANSAS BCDDP STRATIFIED BY MODALITY FINDINGS AND AGE AT SURGERY

Suspicious Modality	*Age at Surgery* <50 Number	<50 Per cent	≥50 Number	≥50 Per cent	Unknown Number	Unknown Per cent	TOTAL Number	TOTAL Per cent
Mammogram only	11	42.3	39	48.8	3	60.0	53	47.7
Mammogram & physical exam	14	53.8	34	42.5	2	40.0	50	45.0
Physical exam only	0	0.0	5	6.3	0	0.0	5	4.5
Unknown	1	3.8	2	2.5	0	0.0	3	2.7
Total	26	100.0	80	100.0	5	100.0	111	100.0

* Includes modalities that have findings with one or more features interpreted as suspicious of malignant or benign disease.

women electing routine mammography. The effect of the change in guidelines was that the dependence between the modalities increased and the opportunity for detection by mammography alone was decreased.

Table VII presents the distribution of modality findings by age at surgery. Lo-dose film mammography was quite effective in detecting cancers of the breast. Mammography was positive in 92.7 per cent of the cancers detected by the Kansas BCDDP. It alone was responsible for the biopsy recommendation in 47.7 per cent of the cancers detected. It was found to be effective in detecting cancers in both younger and older women. In women under age 50 years, mammography was positive in 96.1 per cent of the cancers detected, and it alone was positive in 42.3 per cent. For women over 50 years of age at surgery, mammography showed suspicious findings in 91.3 per cent of the cancer cases, and was the only positive modality in 48.8 per cent of cases. This is not to say that mammography is the only effective modality in annual breast cancer screening; physical examination is obviously very important. Physical examination was positive in nearly 50 per cent of cancers detected, and 4.5 per cent of cases were found by physical examination alone. It should be stressed that the modalities are complementary to one another, and an effective screening program should include deliberate medical correlation of all modality findings.

Analysis of suspicious modality findings and breast cancer lesion size is presented in *Table VIII*. This table emphasizes the relative importance of mammography in detecting small breast cancers in the Kansas program. Of the noninfiltrating breast cancers, 73.3 per cent were detected by mammography alone, and another 20.0 per cent were detected by both mammography and physical examination. In the infiltrating cancers less than 1 cm, again mammography played an important role. Mammography

TABLE VIII
BREAST CANCERS DETECTED BY THE KANSAS BCDDP STRATIFIED BY LESION SIZE AND MODALITY FINDINGS

Suspicious Modality	*Lesion Size* *Noninfiltrating Breast Cancers* Number	Per cent	*Infiltrating Breast Cancers <1cm* Number	Per cent	*Infiltrating Breast Cancers ≥1cm* Number	Per cent	*Breast Cancer Size Not specified* Number	Per cent	*Total* Number	Per cent
Mammogram only	11	73.3	8	61.5	31	40.3	3	50.0	53	47.7
Mammogram & physical exam	3	20.0	4	30.8	40	51.9	3	50.0	50	45.0
Physical exam only	1	6.7	0	0.0	4	5.2	0	0.0	5	4.5
Unknown	0	0.0	1	7.7	2	2.6	0	0.0	3	2.7
Total	15	100.0	13	100.0	77	100.0	6	100.0	111	100.0

TABLE IX
BREAST CANCERS DETECTED BY THE KANSAS BCDDP STRATIFIED BY LESION SIZE AND NODAL STATUS AT SURGERY

Nodal Status at Surgery	Lesion Size: Noninfiltrating Breast Cancers Number	Per cent	Infiltrating Breast Cancers <1cm Number	Per cent	Infiltrating Breast Cancers ≥1cm Number	Per cent	Breast Cancer Size Not specified Number	Per cent	Total Number	Per cent
Positive	0	0.0	0	0.0	26	33.8	0	0.0	26	23.4
Negative	10	66.7	11	84.6	46	59.7	4	66.7	71	64.0
No Nodes Examined	4	26.7	2	15.4	5	6.5	0	0.0	11	9.9
Data NA	1	6.7	0	0.0	0	0.0	2	33.3	3	2.7
Total	15	100.0	13	100.0	77	100.0	6	100.0	111	100.0

alone was responsible for the biopsy recommendation in 61.5 per cent of those detected, and was positive in 92.3 per cent of cases. Physical examination also detected some small cancers in the absence of positive mammography findings, but to a much smaller degree than mammography alone.

Table IX presents the stratification of breast cancer lesion size by nodal status at surgery. Since nodal status at surgery is a good indicator of prognosis, it is important to examine the extent to which routine screening affects nodal status. Only 23.4 per cent of the cancers detected by the Kansas BCDDP had positive nodes at surgery. This is compared with reports from outside screening programs where 53 per cent of all breast cancer cases present with positive nodes.[10] As expected, the noninfiltrating breast cancers either did not have any positive nodes, or did not have findings warranting nodal dissection. No positive nodes were detected in the infiltrating breast cancers smaller than 1 cm, and only 33.8 per cent of those greater than 1 cm had positive nodal status at surgery. These data suggest that routine screening results in the earlier detection of breast cancers which presumably improves prognosis.

Evaluation

The results of the Kansas BCDDP show that it is possible to successfully screen asymptomatic women for breast cancer with physical examination and mammography on an annual basis. The program detected a high percentage of early stage breast cancers without nodal involvement at surgery which favorably affects prognosis and mortality. However, these are not the only criteria by which to evaluate the program. The Kansas BCDDP's performance compared with the other 28 centers of the BCDDP *(Table X)* is also important. One-to-one comparison of the projects is difficult because age distribution and consequent prevalence of breast disease in the populations screened vary among the centers. The emphasis of the BCDDP was on "demonstration" and the projects were designed to meet the needs of their local communities. Consequently, when the research protocol was implemented the projects varied in terms of screening center design and operation. In relation to the BCDDP as a whole, the Kansas center fared well and the cancer detection rates are comparable to those of all centers combined. The biopsy rates, cancer detection rates, minimal cancer detection rates, and interval cancer rates are all slightly lower than the comparable rates for all centers combined, reflecting the younger age distribution of the population screened in Kansas. The percentage of cancers detected by mammography alone in these younger women are higher than for all centers combined, and the per cent of positive nodes in these smaller cancers (noninfiltrating or infiltrating <1cm) was lower.

Other criteria by which to evaluate the Kansas

TABLE X
COMPARISON OF THE KANSAS BCDDP WITH ALL 29 BCDDP CENTERS COMBINED

Rates*	Kansas BCDDP	Entire BCDDP
Biopsy rates	157.2	210.3
Cancer detection rates	28.7	33.1
Minimal cancer detection rates	7.3	10.7
Interval cancer rates	5.2	6.9

* Rates are per 10,000 annual screenings.

TABLE XI
BENIGN BIOPSY RATES

Year	Benign Biopsies per 1,000 Annual Screenings	
	Number	Rate
1	248	24.9
2	98	11.8
3	106	14.1
4	51	7.6
5	20	3.4

TABLE XII
PREDICTIVE VALUE OF BIOPSIES TO DETECT BREAST CANCER

Year	Cancers Detected per 100 Biopsies Performed	
	Number	Rate
1	57	18.9
2	19	17.1
3	11	10.1
4	15	25.0
5	9	36.0

BCDDP relate to the way the program responded to perceived difficulties in implementing breast cancer screening programs in general. One area of concern was the use of nurses to perform physical examinations. From the outset the Kansas BCDDP utilized specially trained nurse clinicians to perform the physical examinations and to teach BSE. The nurses were trained by qualified physicians who also provided quality control and back up by performing random repeat examinations and by being available for consultation at all times.

The data suggest that as the nurses gained more experience in performing physical examinations fewer women with benign conditions were referred for surgical procedures. The rate of benign biopsies decreased and the predictive value positive of biopsies to detect cancer increased. The benign biopsy rate was 24.4/1000 annual screenings in year one and fell to 3.4/1000 annual screenings in year five. The predictive value positive ranged from 18.9 per cent in year one to 36.0 per cent in year five *(Tables XI* and *XII)*. In general, a doubting medical community was persuaded that nurse clinicians could perform highly competent physical examinations of the breast. To provide as comfortable an atmosphere as possible for the all-female population to be screened, the nurse examiners chosen for this project were all women. The response of participants toward these nurse examiners was quite favorable. Many women remarked that they were pleased with being examined by a woman, and thought the examinations were quite thorough. Another advantage gained was the ability of nurse clinicians to elicit questions from screenees and their patience in responding to any questions or concerns.

A second area of concern that the BCDDP had to address was that of risks related to radiation exposure. In 1977, the news that there was an association between radiation exposure and an increased incidence of breast cancer caused a "mammography scare" across the country. One of the immediate consequences for the Kansas BCDDP was a significant decrease in the number of women electing to be screened with mammography. As mentioned previously, eligibility for women under 50 years of age was restricted to those at high risk. Prior to the "scare," the Kansas BCDDP had already successfully lowered the radiation dose of a film-screen mammogram from a skin exposure of 14.1-26.7 R/view in 1973 to less than one Roentgen *(Table XIII)* and newer technology now allows even lower radiation exposures (0.18-0.48 R). The nurse clinicians and physicians implemented a campaign to reeducate participants about the benefits of lo-dose mammography and to rebuild their confidence in the screening program.

(Continued on page 165)

TABLE XIII
REDUCTION IN RADIATION EXPOSURE UTILIZING LO-DOSE FILM — SCREEN SYSTEM

		Phototimer Setting	Skin Exposure*
1973	Kodak RP/M film 35 kVp 35mA	7.6	14.1 - 26.7 R
1973-1974	Kodak XG system 35 kVp 35 mA	4.5	0.9 - 1.4 R
1974-1976	DuPont Lo Do system 35 kVp 35 mA	4.0	0.9 - 1.2 R
1976-1978	Kodak MIN-R system 35 kVp 35 mA	3.0	0.3 - 0.8 R
1978-1982	Kodak ORTHO-M system 35 kVp 10 mA	2.0	0.18 - 0.48 R

* Room 1: C.G.R. Senograph, Molybdenum target, focal spot 0.8 x 1.1 mm, 0.03 Mo filter, water cooled; Room 2: C.G.R. Senograph, Molybdenum target, focal spot 0.8 x 1.1 mm., 0.03 Mo filter, water cooled.

Postpartum Psychiatric Disorders

A Review of Patients at UKSM-KC

LAURA M. BICHEL *and* CHARLES R. KING, M.D., *Kansas City, Kansas*

POSTPARTUM psychiatric disorders include a broad range of emotional problems generally occurring within three months after parturition. The disturbance may be a transient event lasting only a few days or weeks or may be months or more in duration. The three general types of postpartum psychiatric reactions are: (1) "blues syndrome" or "baby blues," a transient self limiting period of emotional lability; (2) postpartum psychosis characterized by schizophrenic, manic, or depressive psychotic symptoms; and (3) chronic postpartum depression of unpredictable severity and duration.

While the etiology of postpartum psychiatric disorders is uncertain, current hypothesis involves a combination of biological events, psychological factors, social events, and interpersonal activities all contributing simultaneously to the emotional vulnerability of the postpartum woman. Biological events include the sudden drop in circulating hormones which occurs in the immediate postpartum period as well as changes in electrolytes, fluid balance, and even sleep patterns. The physical stress and work of labor and delivery may also contribute to the development of the emotional vulnerability seen in the postpartum patient. Pre-existing marital or psychological problems, conflicts regarding motherhood, the role and fears of failure as a mother all predispose to the development of postpartum psychiatric disorders. Patients with pregnancy complications, both physical and emotional, have a higher incidence of postpartum psychiatric disorder. A family history of psychiatric difficulty is more common among patients with postpartum disease.

Methods

All obstetrical patients who delivered at the University of Kansas Medical Center during the period of January 1972 through March 1982 were reviewed. From this total of 14,652 patients, seven were diagnosed with a postpartum psychiatric disorder. Information was gathered regarding age, race, obstetrical history, pregnancy complications, delivery complications, method of delivery, and hospital stay following delivery. The onset of symptoms following delivery was noted as well as the symptoms themselves, type of treatment, hospital stay, and diagnosis of the puerperal psychiatric reaction. Information concerning previous psychiatric problems and family history of psychiatric problems was also recorded. Previous psychiatric disorders were defined as any psychiatric hospitalization with a discharge diagnosis of mental illness other than drug or alcohol abuse. A positive family history of psychiatric problems included all general types of mental illness requiring psychiatric treatment.

From the Department of Obstetrics and Gynecology, Division of Reproductive Genetics and Perinatology, The University of Kansas School of Medicine-Kansas City.

There are three distinct types of postpartum psychiatric disturbance, all likely to begin within two months following delivery. Results of this review generally confirm the conclusions of prior studies.

Case Summaries

Case One: A 20-year-old black female ($G_2P_1LC_1TAB_1$) with no previous history of psychiatric problems delivered a full term viable female infant by cesarean section. Her postpartum course was complicated only by a febrile episode successfully treated with antibiotics. She was discharged eight days after delivery. The patient was brought to the emergency room on the ninth postpartum day by her family because of agitation with uncooperative and bizarre behavior. Mental status examination identified circumstantiality of speech, looseness of associations, echolalia, auditory hallucinations, and delusions concerning the baby. She had no suicidal or homicidal ideation and was well oriented. Following admission to the Psychiatry service, therapy with Haldol, Navane, and Artane was started. The patient improved, and within a few days was exhibiting no abnormal behavior, speech, or thought disorders. She was discharged on the 13th hospital day with a diagnosis of postpartum psychosis.

Case Two: A 22-year-old white female ($G_2P_1LC_1$) with no history of previous psychiatric disturbance was delivered of a full term viable male infant by spontaneous vaginal delivery. The patient became psychotic during the delivery and demanded that she be allowed to go home and see her other baby who had actually died at three months of age. Following the delivery, the patient became very hostile, refused to acknowledge the new baby, stated she did not want it, and was very preoccupied with thoughts and demands concerning her dead baby. The patient was given Valium intravenously. The next day the patient was calm, cooperative, and receptive to the infant. Psychiatric consultation was obtained and a diagnosis of postpartum psychosis and depression was made. Outpatient psychiatric treatment was initiated following discharge on the third postpartum day.

Case Three: A 24-year-old white female ($G_1P_1LC_1$) with no previous history of psychiatric disorder had a premature spontaneous vaginal delivery of twin boys. The patient had no postpartum complications and was routinely discharged. The infants remained hospitalized. Eight days following delivery the patient was admitted to the Psychiatry service with complaints of inability to sleep for three days, continual talking, reading of the Bible, praying, and hearing God's voice talking to her. On mental status examination she was noted to have delusions of grandeur, auditory hallucinations, and inappropriate and labile affect. She had no suicidal or homicidal ideation, and was well oriented. She was treated with Thorazine. After three days her behavior was appropriate and she became anxious to be discharged. The patient left the hospital against medical advice with a diagnosis of postpartum psychosis. She subsequently was diagnosed as having manic-depressive illness.

Case Four: A 23-year-old white female ($G_2P_2LC_2$) was delivered of a full term viable male infant by spontaneous vaginal delivery. This patient had a history of multiple episodes of drug abuse but no other psychiatric problems. She presented to the emergency room approximately six months after delivery with a chief complaint of not knowing whether she was a man or a woman, and of feeling sinful, dirty, and unworthy. She stated that the symptoms commenced soon after delivery and that they had worsened during the ensuing months. She also noted weight loss of about ten pounds and hypersomnia during the preceding months. The patient's mental status examination revealed delayed speech, tangentiality of speech, delusions, depressed mood and affect, and suicidal thoughts but no suicidal plans. She was admitted to the Psychiatry service, and therapy with Navane, nortriptyline, and Artane was started. Significant family problems were identified, and as a result family counselling was started. The patient improved gradually and was discharged on the 26th day of hospitalization with a diagnosis of postpartum depression. This patient continued to have recurrent depressive episodes in spite of therapy, and ultimately was committed to a state mental hospital seven months after discharge.

Case Five: A 39-year-old black female ($G_6P_6LC_6$) with a previous psychiatric diagnosis of paranoid schizophrenia was delivered of a full term female infant, her sixth child, by spontaneous vaginal delivery. Following delivery the patient pulled out her intravenous apparatus and began running up and down the halls screaming that the nurses were trying to kill her. Therapy with Thorazine, Prolixin, and Artane was started. The patient progressed slowly and was discharged 64 days after delivery. Her diagnosis was mental retardation, paranoid schizophrenia, and postpartum psychosis. Two years earlier following the birth of her fifth child the patient had required psychiatric admission.

Case Six: An 18-year-old black female ($G_2P_1LC_1TAB_1$) with history of previous psychiatric admissions for drug abuse and mental retardation was delivered of a viable full term infant by cesarean section. Delivery was followed by an elective hysterectomy. Eight days following discharge the patient was brought to the emergency room by her family because of uncooperativeness, agitation, and bizarre behavior. Mental status examination showed her to be uncooperative, with decreased spontaneity of speech, blunted affect, and an anxious, excited mood. She did not have delusions, but was found to have mild tactile hallucinations. Her intelligence was judged to be retarded. Therapy with Haldol and Artane was started. The patient's hospitalization was complicated by a primary infection with cytomegalovirus. Following this febrile illness, the patient slowly progressed and improved. She was granted a weekend pass from which she never returned. She was discharged against medical advice with a diagnosis of chronic schizoaffective disorder, postpartum psychosis in remission, and mental retardation.

Case seven: A 29-year-old white female ($G_2P_2LC_2$) with no previous history of psychiatric disorder was delivered of a viable full term infant by spontaneous vaginal delivery six weeks prior to admission. The patient was brought to the emergency room by her husband who stated that she had been disoriented and had "not been acting quite right" for several days. This abnormal behavior included not

TABLE I
PATIENT SUMMARY

Case	*Age (Years)*	*Previous Psychiatric History*	*Family Psychiatric History*	*Pregnancy Complications*	*Delivery*	*Postpartum Complications*	*Onset Symptoms (Days)*	*Initial Symptom*	*Psychiatric Hospitalization (Days)*	*Therapy*	*Diagnosis*
1	20	–	+	–	C-section	Febrile morbidity	9	Agitation, delusions, bizarre behavior	13	Haldol Navane Artane	Postpartum psychosis
2	22	–	–	Gestational diabetes, hypoglycemia	Spontaneous	Psychotic with delivery	At delivery	Delusions, acute psychosis	4	Valium	Postpartum psychosis, depression
3	24	–	+	Premature, twins	Spontaneous	–	8	Insomnia, hallucination	4	Thorazine	Postpartum psychosis, manic-depressive illness
4	23	–	–	–	Spontaneous	–	14	Delusions, depression	26	Navane, nortriptyline, Artane	Postpartum depression
5	39	+	+	Hypertension	Spontaneous	–	At delivery	Delusions, inappropriate behavior	64	Thorazine, Prolixin, Artane	Postpartum psychosis, paranoid schizophrenic
6	18	+	?	–	C-section	Hysterectomy	8	Bizarre behavior, hallucination	74	Haldol, Artane	Postpartum psychosis, chronic schizo-affective disorder
7	29	–	–	–	Spontaneous	–	5	Hallucination, delusion, insomnia	10	Haldol, Benadryl	Postpartum psychosis

sleeping for two nights, not taking proper care of the baby, and complaining of hearing voices speaking to her about God and her children, and telling her that she was going to die. In addition, the patient complained of fever and chills for four days prior to admission, and numbness and tingling in her left arm for two days. During the initial examination the patient was found to have a full affect, depressed mood, auditory hallucinations, delusions and decreased responsiveness, but no suicidal or homicidal ideation. During the initial evaluation of this patient she became unresponsive and her behavior was catatonic in nature. Therapy with Haldol and Benadryl was started. During the next few days the patiient's psychosis began to clear although she continued to have auditory hallucinations. She was discharged on the tenth hospital day to be followed as a psychiatric outpatient. Her discharge diagnosis was probably postpartum psychosis.

Results and Discussion

Most postpartum psychiatric disturbances begin in the first six to eight weeks following delivery. The "baby blues" has an onset within the first week after delivery. It is seen in 50-80 per cent of "normal" postpartum women. It rarely lasts longer than ten days and is usually self limiting. Symptoms include episodic crying spells, rapid mood swings, sleep disturbances, and heightened sensitivity to interactions with others particularly to feelings of "supposed rejection." Characteristically no disorientation, personality disorganization, memory, or attention deficits are seen.

Postpartum psychosis is a major postpartum psychiatric disorder which is diagnosed in one or two patients/1000 births, with a higher incidence in primiparas. The incidence of one in 2,000 pregnancies in this series probably represents under reporting of the condition since only patients actually hospitalized were included in this study. Increased vulnerability exists during the first three months of the puerperium with 50 per cent of patients presenting during the first two weeks. There is often an exaggeration or continuation of the "baby blues" symptoms for these women; however symptoms progress to those of acute schizophrenia, a major affective disorder (manic or depressive), or a severe chronic unipolar depression. These mothers most often require hospitalization and medication with antipsychotics or lithium carbonate. Ninety per cent of these women are ultimately able to return to their families and function at their pre-hospitalization level. Patients with a previous diagnosis of a bipolar affective disorder or chronic schizophrenia have a much poorer prognosis.

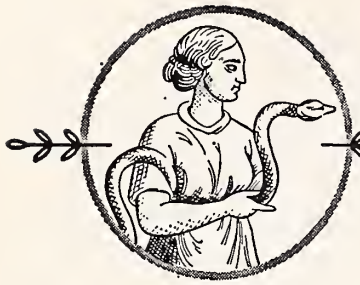
The third type of postpartum psychiatric disturbance is that of severe, chronic depression. Usually onset is in the early postpartum months characteristically with a slow and insidious latent period of several weeks. Severe postpartum depression occurs in 10-20 per cent of postpartum patients and may persist for a year or more. Symptoms are those typical of depression — altered sleep patterns, lack of interest in usual activities, difficulty concentrating, and disturbances of thought processes. Patients often require hospitalization, psychotherapy, and treatment with antidepressive medication. Electroconvulsive therapy may be indicated if medication alone is unsuccessful. The prognosis of these patients is quite unpredictable.

In general the patients from this study demonstrated conditions that have been reported in the literature to predispose to postpartum psychosis. A history of previous psychiatric disturbances in the patient, a positive family history of psychiatric illness, and complications during pregnancy are particularly noteworthy in this regard *(Table I)*. Two of seven patients in this study had a history of previous psychiatric disorders. Forty-three per cent of patients had a positive family history of psychiatric disease, and a similar number also experienced complications during pregnancy. In contrast to the current literature, which indicates that the majority of patients who develop postpartum psychoses are primiparas, the patients identified in this study were all multiparous, except for one patient experiencing her first pregnancy. Twenty-two per cent of patients had had a previous pregnancy termination.

The onset of postpartum psychiatric symptoms is generally early in the puerperium. Psychosis usually appears during the first two weeks, whereas depression most often has an insidious onset with symptoms recognized only later. All patients in this study had onset of symptoms within the first two weeks postpartum with the exception of one patient whose actual onset was indefinite. Four patients in this series recovered within two weeks (two within four days) and the other three patients required as long as ten weeks. The majority of patients with prolonged recovery had a history of previous psychiatric disease.

Characteristic symptoms of psychosis such as agitation, emotional lability, restlessness, auditory hallucinations, and delusions were observed in our patients. Abnormalities of speech such as disorganization, loose associations, and lack of goal directedness were also noted. However, no impair-

(Continued on page 162)



Medical HISTORY

Family Medicine Education

A Kansas Historical Review, 1967-1982

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KANSAS has long been recognized as a national leader in Family Medicine Education at the post-MD level, beginning with the establishment of one of the first family practice residency training programs in the country at Wesley Medical Center in Wichita in 1967 and culminating with the establishment of a fifth training program at the Smoky Hill Family Practice Center in Salina in 1979.

This paper reviews the historical development of the five Family Practice residency programs, the track record in terms of the graduates from the programs, the contribution to undergraduate health professional education, and the impact of the programs on health services to Kansans.

Wesley Residency

The Wesley Family Practice Residency Program in Wichita was begun in 1967 by G. Gayle Stephens, M.D. In 1968, Dr. Stephens developed a model ambulatory family practice center, drawing from his own private patients. In 1969, the specialty of Family Practice was recognized for the first time with the formation of the American Board of Family Practice. That same year, a residency review committee for the specialty met for the first time and accredited five programs, among them the Wesley program. Dr. Stephens, later to become president of the Society of Teachers of Family Medicine, saw the Wesley model, with few modifications, accepted as the national standard for family practice graduate education.

In 1972, Stanley Mosier, M.D. and Victor Vorhees, M.D., both 1971 graduates of the Wesley program, became co-directors. Drs. Mosier and Vorhees at that time became the first residency trained family physicians in the country to direct a family practice residency program. In 1980, David M. Holden, M.D. became Director of the program.

The five Family Practice Residency Programs in Kansas augment the health care delivery systems in several Kansas communities. These programs have combined to produce a total of 233 graduates, 135 of whom now practice family medicine in Kansas.

In 1980, the Wesley program became an integral part of the Department of Family and Community Medicine at the University of Kansas School of Medicine-Wichita. The proper name today is the Wesley/UKSM-Wichita Family Practice Residency Program, and all faculty hold full-time appointments to the medical school and participate in teaching undergraduate students during the Family and Community Medicine clerkships.

Eighty-one family physicians have graduated from the Wesley program and 49 of them (60%) are practicing in Kansas. Currently there are 24 residents in training. In order to provide the educational base for the residency training program, the Wesley program utilizes two ambulatory clinics and admits

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to the Wesley Medical Center Hospital. Approximately 20,000 ambulatory visits, 480 hospital admissions, and 300 obstetrical deliveries (and newborns) comprise the annual contribution to the health care delivery system for the Wichita area.

The Wesley Family Practice residency program participates in several areas of health professional education in addition to post-MD training including third and fourth year medical students on clinical clerkships from UKSM-Wichita, nurse clinician students from Wichita State University, and dietetic students from Kansas State University.

St. Joseph Residency

In June, 1970, the Residency Review Committee for Family Practice granted provisional approval to the St. Joseph Hospital and Rehabilitation Center, Wichita, for a Family Practice Residency Program, making it the 44th Family Practice Residency Program in the United States. At that time, Lew W. Purinton, M.D., Director for Medical Education and Robert Purvis, M.D., Assistant Director for Medical Education were the responsible persons for the residency program in the absence of a named Director.

James M. Donnell, M.D. was named the first residency program director in 1971. Subsequent directors have been Dr. Purinton — Interim Director, 1976-77; Lawrence Miller, M.D., 1977-79; and R. E. Riederer, M.D., 1979-present.

Fifty-six family physicians have completed the training program at St. Joseph Medical Center and 25 (45%) are practicing in Kansas. Currently there are 22 residents in training.

St. Joseph's Family Practice Ambulatory Center was opened in December 1971, has been enlarged twice, and is now scheduled for a third enlargement. Planning is underway for a 3,600 square-foot addition to be built in 1983. This addition is needed for increasing out-patient care activity and full-time teaching staff with a portion of their time in patient care. Approximately 28,000 ambulatory visits, 1800 hospital admissions, and 400 obstetrical deliveries (and newborns) annually form the educational basis for this residency teaching program.

The Family Practice Residency Program became affiliated with UKSM-Wichita in 1975. This affiliation has been strengthened with the development of a management contract with the school effective July 1, 1982. This contract calls for input into the goals, objectives, faculty selection, curricular development, and overall program direction. The program remains under day-to-day management by its staff, which is funded by St. Joseph Medical Center. The program name has been changed to St. Joseph Medical Center Family Practice/UKSM-Wichita Residency.

The Residency has provided an elective rotation for fourth year medical students. Student nurses from Wichita State University and St. Mary's of the Plains Nursing Schools have had office experience with the Center. Nurse Clinicians have had portions of their training there.

St. Francis Residency

The St. Francis Regional Medical Center Family Practice Residency, Wichita, was established in 1975 and had its first resident trainees in July 1976. Donald J. Gessler, M.D. became the initial full-time Program Director on April 1, 1975, and continues as the present Director.

The Program has graduated 23 family physicians and 19 (82%) are practicing in Kansas. Currently there are 16 residents in training.

The 10,000 square foot Family Practice Center is housed in a remodeled nursing home approximately 1½ blocks north of the main hospital complex. The clinical population for the residency teaching program comes from approximately 12,000 ambulatory visits, 650 admissions to St. Francis Regional Medical Center Hospital, and 284 obstetrical deliveries (and newborns). In addition, the residents provide medical services to the Parallax Drug Program, hold two Family Practice clinics per week at the Indian Health Service Clinic, and provide occupational medical services for a local packing company. The residents also are involved on a part-time basis in providing health care for various high school sports activities and other civic activities as requested. Dietetic students from Kansas State University receive clinical experience with the residency program, and Medical Assistant students from the Bryan Institute also train there on a part-time basis.

The residency program at St. Francis is not under the administrative control of UKSM-Wichita; however it does have an educational affiliation.

Medical students in a required Family Practice rotation from UKSM-Wichita are with the residency training program six months of each year.

UKSM-KC Residency

The Family Practice Residency Program at the University of Kansas Medical Center in Kansas City was established in 1971 and received formal approval in early 1972. The first three resident trainees began on July 1, 1972. Jack Walker, M.D. was the first Director of the program and Chairman of the Department of Family Practice at the Medical Center. Dr. Walker served in that capacity until June 30,

1982. James G. Price, M.D. was recently named Chairman of the Department and Director of the Residency Program.

Seventy family physicians have completed the program and 40 (57%) are practicing in Kansas. Currently there are 36 residents in training.

For a number of years the Kansas University Medical Center program operated two Family Practice Ambulatory Clinics to provide the educational base for residency training. In March 1982, the two clinics were combined into a single ambulatory clinic located on the ground floor of the new Bell Memorial Hospital. Approximately 30,000 ambulatory visits, 445 admissions to the hospital, and 188 obstetrical deliveries (and newborns) annually contribute to the health care delivery system for the Metropolitan Kansas City area.

The program at Kansas University Medical Center contributes to several areas of health professional education in addition to resident training. Faculty and residents participate in several undergraduate medical student courses including the first year Introduction to the Clinical Process, the second year Physical Diagnosis course, and a fourth year Family Practice elective. Graduate students in medical social work and graduate nursing students receive a part of their clinical experience with the Family Practice Center.

Smoky Hill Residency

The residency training program in Salina is the newest of the five programs in Kansas. It is unique in that it is the only residency program that was actually created by an act of the Kansas Legislature.

With the passage of S.B. 472, the 1977 Legislature established as a state policy the promotion of family practice residency programs at locations in the state other than Kansas City and Wichita. The programs are formalized through affiliation agreements between UKSM and qualified medical care facilities or non-profit community operations. The enabling legislation authorized the School of Medicine to enter into such agreements. The act prohibited entering into such agreements in any county with a population greater than 200,000.

Negotiations with interested groups in Salina led to the eventual signing of an affiliation agreement effective July 1, 1978, with the Salina Health Education Foundation (SHEF) for operation of the Smoky Hill Family Practice Program. The foundation is a nonprofit corporation whose membership includes all physicians in the Saline County Medical Society (approximately 80). The foundation is governed by an 11-member executive board. The Board employed Robert Brown, M.D. as executive secretary and medical education director, and Acting Director of the Family Practice Residency Program. Apart from its principal responsibility for conducting the family practice residency training program, SHEF also sponsors continuing medical education programs, supports and encourages development of medical library resources, and provides some coordination of medical preceptorships and other residency rotations in Salina.

The residency program is under the administrative direction of the Department of Family and Community Medicine at UKSM-Wichita.

The residency program is an affiliated program with the St. Joseph Family Practice Residency in Wichita. The first year takes place in Wichita, and

TABLE I
CURRENT RESIDENT PHYSICIANS IN TRAINING*

Program	*R-1*	*R-2*	*R-3*	*Total*
Wesley	8	6	10	24
KUMC	12	13	11	36
St. Joseph	8	7	7	22
St. Francis	6	5	5	16
Smoky Hill	4†	4	2	10
Total	38	35	35	108

* July 1, 1982.
† (at St. Joseph)

TABLE II
APPROXIMATE CLINICAL HEALTH SERVICES*

Program	*Ambulatory Visits*	*Hospital Admissions*	*Obstetrical Deliveries*
Wesley	20,000	480	300
KUMC	30,000	445	188
St. Joseph	28,000	1,800	400
St. Francis	12,000	650	284
Smoky Hill (Salina)	6,400	250	50
Total	96,400	3,625	1,222

* July 1, 1981-June 30, 1982.

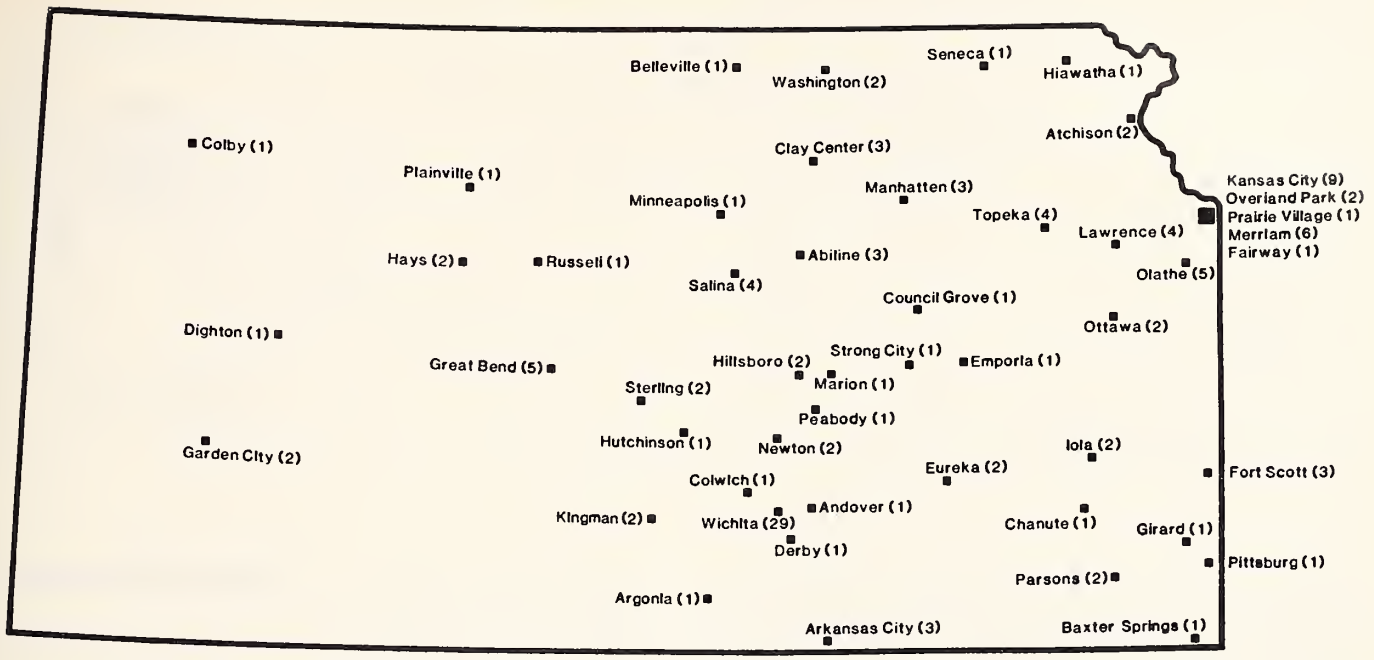


Figure 1. The locations of graduates of Kansas Family Practice Residency Programs in Kansas are shown above. This includes only family physicians who completed training in the five residency training programs in Kansas.

the second and third years of training take place in Salina.

The Smoky Hill Family Practice Program got under way in the summer of 1979 with the admission of three first-year residents into St. Joseph's residency training program.

The program graduated its first three in June 1982, and two (66%) are practicing in Kansas. Currently there are ten residents in training — four in the first year in Wichita, and six in the second and third years in Salina.

On September 1, 1982, Timothy Scanlan, M.D. was appointed Program Director for the Smoky Hill Family Practice Program.

The residency program is based in the Smoky Hill Family Practice Center located on the campus of Kansas Wesleyan University, and the residents receive training and admit patients to St. John's Hospital and Asbury Hospital in Salina.

Forming an educational base for the second and third years of the residency are approximately 6,400 ambulatory patients, 250 hospital admissions, and 50 obstetrical deliveries annually.

The residents participate in the operation of the drug and alcohol detoxification unit at St. John's Hospital, and plans are being developed for participation of residents in the emergency medical services of the two local hospitals. Residents are assigned two afternoons per month to the City/County Health Department where they participate in the care of obstetrical and gynecological patients; they also see patients at local nursing homes on a regular basis.

Summary

The distribution of residents in training at the five programs as of July 1982 is shown in *Table I*. *Table II* defines the clinical services provided by each of the residency programs from July 1, 1981 to June 30, 1982.

Graduates of the five Family Practice Residency Programs in Kansas are producing a significant impact upon the supply of family physicians for Kansas. One hundred thirty-five (57%) of the 233 graduates are practicing in Kansas *(Table III)*.

Graduates of the five programs are now practicing and teaching family medicine in 50 Kansas communities *(Figure 1)*. The survey includes only family physicians who are graduates of the residency training programs in Kansas. A number of other residency trained family physicians who received their training in programs outside of Kansas are also practicing in Kansas communities.

In addition to the primary educational mission of training family physicians, the residency programs contribute significantly to the health care delivery system for Kansas citizens *(Table II)*. The programs contribute to the undergraduate education of medical students and other health professional students. The

TABLE III
LOCATION OF GRADUATES BY STATES*

Name of Program	*Number of Graduates*	*Kansas*	*Missouri*	*Oklahoma*	*Nebraska*	*Colorado*	*Other*
Wesley	81	49	3	4	3	6	16
KUMC	70	40	9	1	0	1	19
St. Joseph	56	25	5	7	3	0	16
St. Francis	23	19	1	0	0	0	3
Smoky Hill	3	2	0	0	0	0	1
Total	233	135	18	12	6	7	55

* Data for November, 1982.

programs provide health services to a sizable number of patients, both outside the hospital and within hospitals. A number of Kansas communities have benefited by having new family physicians located in their areas. A continuous cadre of new family physicians will annually emerge from the programs.

The programs have both a direct and indirect — immediate and future — impact upon the economy of the State of Kansas.

The national movement to educate more family physicians which began in the late 1960s is paying off in Kansas, and the five Family Practice Residency Programs based in Kansas are making a significant contribution.

CORRECTIONS

The article entitled, "Nausea and Vomiting: Its Management in Cancer Chemotherapy," by Roy H. Hart, M.D., published in the January issue of the *Journal,* contains an error. On page 13, left hand column, the final sentence should read as follows: "Short-term EPS, which excludes the long-term complication of tardive dyskinesia, seldom looms as a problem to the neuropsychiatrist, since there is a wide enough selection of drugs to control it."

The Drug Information section was inadvertently omitted from the contents in the January issue. That section, presenting a discussion of isotretinoin (Accutane), may be found on page 30.

The *Journal* sincerely regrets these errors and any resulting confusion.



Anesthesia

A History of Simpson's Discovery of Chloroform

SUSAN CRIDER, M.D., *New Brunswick, New Jersey*

H. Laing Gordon, in 1897, wrote of Simpson and the practice of surgery:

The surroundings of the surgeon at the commencement of the century were vastly more repugnant to a youth of sensitive nature than today. The operating theatre then has been compared to a butcher's shambles; cleanliness was not considered necessary, and little attention was paid to the feelings of the patient. He was held down by three or four pairs of powerful arms as the surgeon boldly and rapidly did his work, despite the screams, stopping, perhaps only to roughly abuse the patient for some agonized movement which had interfered with the course of action. The poor wretch saw the instruments handed one by one by the assistant, and heard the surgeon's calm directions and his remarks on the case. The barbarous practice of arresting bleeding by the application of red-hot irons to the surface of the wound had indeed ceased three centuries before, when that humane reformer, Paré, displaced it with the method of tying the open blood vessel, but the patient's blood gushed forth before him until arrested, into the sawdust spread to receive it, and the sight and the hot odour of it oftentimes mercifully caused him to faint. . . . When Simpson first saw Liston raise his knife to operate on a poor Highland woman, he actually felt so repelled that he contemplated abandoning his studies, and made a serious attempt to enter upon legal work instead. But the mental struggle with which medical men of all countries, and in all times, can sympathise out of their own knowledge, ended in a victory for medicine, and a triumphant return to his studies with the question permanently engraved on the tablets of his mind, "Can nothing be done to prevent this suffering?"[1]

Credit for development of anesthesia must rightfully be shared by those who discovered the agents and those who overcame the opposition to its use. Sir James Y. Simpson of Edinburgh not only discovered chloroform, but also worked diligently for the general acceptance of anesthetic agents.

James Y. Simpson was born in Bathgate, in Linlithgowshire, on June 7, 1811, the seventh son in a family of eight children. The custom at that time was to educate the youngest son to be a scholar. All the children received a good education in the parish schools, but after they had taken advantage of all the education offered in the area they went on to marry, farm, or establish whatever trade they wanted. As the youngest son, James Simpson was very bright and studious. His lifelong interests in archeology, theology, and medicine began then. The libraries in homes at that time consisted of the Bible and religious works and a few pamphlets.

At age 14 years, Simpson entered Edinburgh University where a friend, John Reid, was also studying. Reid later became Professor of Anatomy in the University of St. Andrews and was instrumental in encouraging Simpson's career and interest in medi-

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cine. In 1830 Simpson became a member of the Royal College of Surgeons at age 19 years. After qualifying for the degree of Doctor of Medicine of Edinburgh University in 1832 with a thesis titled "De Causa Mortis in Quibusdam Inflammationibus Proxima," he started work for Professor John Thomson, a pathologist.

If to MacArthur and John Reid was due the credit of first directing Simpson's thoughts to the study of medicine, to Professor John Thomson belongs the credit of having made him an obstetrician. "At Dr. Thomson's earnest suggestion and advice," says Simpson, "I first turned more especially to the study of midwifery with the view of becoming a teacher of this department of medical science."[1]

Simpson applied for the Edinburgh Chair of Midwifery at age 28 years and started one of many controversies he would be involved in during his lifetime.

The objection of his youth was less easily got over than that of his unmarried state. With characteristic promptness, as soon as he had determined to apply for the Chair and found that as a bachelor his chances would be small, he disappeared for a time from Edinburgh, and returned triumphantly with Miss Jessie Grindlay, of Liverpool, as his wife. It was a bold stroke which delighted his supporters, discomfited his opponents, who saw therein the removal of a barrier to his success and a weapon from their hands, and astonished the worthy town councillors in whose gift the appointment lay.[1]

Simpson won the Chair by a vote of 17 to 16.

During his obstetrical practice Simpson researched the many methods that had been tried to prevent the pain of surgery. Sir Humphrey Davy in 1800 had written, "As nitrous oxide in its extensive operations appears capable of destroying physical pain, it may probably be used with advantage during surgical operations in which no great effusion of blood takes place."[2]

On Dec. 10, 1844, Horace Wells was present at a demonstration in which G. Q. Colton, a chemist, prepared nitrous oxide. Wells was impressed when he saw a man under the influence of the gas hit his arm on a table with no pain. The next day, under the influence of nitrous oxide, he allowed a student, John Riggs, to extract a tooth from his mouth. This is reported to have been the first anesthetic operation in America. Unfortunately a public demonstration by Wells and a student, William Morton, failed due to premature withdrawal of the bag containing the gas.

Wells and Morton were ignominiously hissed by the crowd of practitioners and students gathered to see the operation. Wells never recovered from the disappointment and the illness which resulted, and although he was able to explain his discovery to the French Academy of Science in 1846, he unfortunately died insane in New York two years later.[1]

It was reported that his illness was caused by self treatment with chloroform, another inhalation anesthetic taking hold in England.

Morton at this time became intrigued by the theory of inhalation anesthetics. In a Memoir to the Academy of Arts and Sciences, Paris (July 1847), Morton admitted that Dr. Charles T. Jackson, a chemist, had called his attention to the sedative effects of ether by inhalation. That was in the summer of 1844, but Morton claimed that he had conceived the idea separately and before Jackson mentioned it to him.

On September 30, 1846, Morton used sulphuric ether to extract a tooth from a Mr. Eben Frost. The following is an account of the first use of sulphuric ether as an anesthetic.

Toward evening a man residing in Boston came in, suffering great pain and wishing to have a tooth extracted. He was afraid of the operation and asked if he could be mesmerized. I told him I had something better, and saturating my handkerchief, gave it to him to inhale. He became unconscious almost immediately. It was dark, and Dr. Hayden held the lamp while I extracted a firmly rooted bicuspid tooth. There was not much alteration in the pulse and no relaxing of the muscles. He recovered in a minute and knew nothing of what had been done to him. He remained for some time talking about the experiment. This was the 30th of September, 1846.[3]

Morton then made arrangements to demonstrate his new anesthetic agent in Boston in the presence of Drs. Warren and Hayward. Dr. Charles T. Jackson was present and credited Morton with the invention of the apparatus for administering ether. Morton was to spend the rest of his life trying to gain a patent for this discovery. He received little in the way of monetary reward for the discovery of sulphuric ether. The controversy over who was the discoverer of anesthesia lasted for years.

On October 17, the patient being prepared for the operation, the apparatus was applied to his mouth by Dr. Morton for about three minutes, at the end of which time he sank into a state of insensibility. I immediately made an incision about three inches long through the skin of the neck, and began a dissection among important nerves and blood vessels without any expression of pain on the patient. Soon after he began to speak incoherently and appeared to be in an agitated state during the remainder of the operation. Being asked immediately afterwards whether he had suffered much, he said that he had felt as if his neck had been scratched; but subsequently, when inquired of by me, his statement was, that he did not experience pain at the time, although aware that the operation was proceeding.[4]

This was an account of the first public demonstration of ether anesthesia.

Although Wells, Morton, and Jackson are rightfully credited with the discovery of anesthesia, Sir James Y. Simpson of Edinburgh was partially responsible for its acceptance and broad use, especially in obstetrics, and for the discovery of chloroform.

A doctor of obstetrics in Edinburgh, Simpson had been testing various agents to find an anesthetic with fewer side effects than sulfuric ether. The discovery of chloroform as an anesthetic was accidental.

> Late one evening, it was the fourth of November, 1847, on returning home after a weary day's labour, Dr. Simpson and his two friends and assistants, Drs. Keith and Duncan, sat down to their somewhat hazardous work in Dr. Simpson's dining-room. Having inhaled several substances, but without much effect, it occurred to Dr. Simpson to try a ponderous material which he had formerly set aside on a lumber-table, and which on account of its great weight he had hitherto regarded as of no likelihood whatever; that happened to be a small bottle of chloroform. It was searched for and recovered from beneath a heap of waste paper. And with each tumbler newly charged, the inhalers resumed their vocation. Immediately an unwonted hilarity seized the party — they became bright-eyed, very happy, and very loquacious — expatiating on the delicious aroma of the new fluid. The conversation was of unusual intelligence, and quite charmed the listeners — some ladies of the family and a naval officer, brother-in-law of Dr. Simpson. But suddenly there was talk of sounds being heard like those of a cotton mill louder and louder; a moment more and then all was quiet — and then crash! On awakening Dr. Simpson's first perception was mental — "This is far stronger and better than ether," said he to himself. His second was to note that he was prostrate on the floor, and that among the friends about him there was both confusion and alarm. Hearing a noise he turned round and saw Dr. Duncan beneath a chair — his jaw dropped, his eyes staring, his head bent half under him; quite unconscious, and snoring in a most determined and alarming manner. More noise still and much motion. And then his eyes overtook Dr. Keith's feet and legs making valorous attempts to overturn the supper table, or more probably to annihilate everything that was on it. By and by, Dr. Simpson having regained his seat, Dr. Duncan having finished his uncomfortable and unrefreshing slumber, and Dr. Keith having come to an arrangement with the table and its contents, the sederunt was resumed. Each expressed himself delighted with this new agent, and its inhalation was repeated many times that night — one of the ladies gallantly taking her place and turn at the table — until the supply of chloroform was fairly exhausted.[3]

The dose of chloroform was much less than that of ether. Its action was found to be quicker and more complete. The odor was much more agreeable and not as irritating to the bronchi. No special apparatus was required.[5]

Simpson wrote:

> I have employed it also in obstetric practice with entire success. The lady to whom it was first exhibited during parturition, had been previously delivered in the country by perforation of the head of the infant, after a labor of three days' duration. In this, her second confinement, pains supervened a fortnight before the full time. Three hours-and-a-half after they commenced, and, ere the first stage of labor was completed, I placed her under the influence of the chloroform, by moistening, with half a teaspoon of the liquid, a pocket handkerchief, rolled up into a funnel shape, and with the broad or open end of the funnel placed over her mouth and nostrils. In consequence of the evaporation of the fluid, it was once more renewed in about ten or twelve minutes. The child was expelled in about 25 minutes after the inhalation was begun. The mother subsequently remained longer soporose than commonly happens after ether. The squalling of the child did not, as usual, rouse her; and some minutes elapsed before the placenta was expelled, and after the child was removed by the nurse into another room, before the patient awoke. She then turned round and observed to me that she had "enjoyed a very comfortable sleep, and indeed required it, as she was so tired, but would now be more able for the work before her." I evaded entering into the conversation with her, believing, as I have already stated, that the most complete possible quietude forms one of the principal secrets for the successful employment of either ether or chloroform. In a little time she again remarked that she was afraid her "sleep had stopped the pains." Shortly afterwards, her infant was brought in by the nurse from the adjoining room, and it was a matter of no small difficulty to convince the astonished mother that the labor was entirely over, and that the child presented to her was really her "own living baby.[5]

Numerous reports were published and chloroform *vs* sulphuric ether became a major controversy. The early reports of chloroform use were positive and few ill effects were observed. Still the legal battles in Boston were raging and people were taking sides over who should be credited with the discovery of anesthesia.

Simpson himself credited Dr. Morton with the discovery of anesthesia. In a letter to Morton on November 19, 1847, he said, "Of course, the great thought is that of producing insensibility to pain; and for that the world is, I think, indebted to you."[6]

Before its toxic effects became known, chloroform became more popular than ether in many areas. In a testimonial A. E. Hosack, February 9, 1848, wrote,

> Chloroform appears to possess many advantages over ether. It is more agreeable to the taste, more rapid in its influence, more composing, more durable in its effects, and to my knowledge has never been attended with any ill consequences; besides, a very small quantity is sufficient to produce the desired effect, while on the contrary, ether

is liable to create considerable excitement, is of shorter duration in its influence, and to many is very disagreeable, and even nauseous; and its after effects are sometimes very alarming, and in a few instances have proved fatal. But chloroform is not entirely free from some of these objections, as it is sometimes quite as exciting as ether, and the patient equally restless during an operation. This effect, however, is not so frequent as with ether. They are both more efficacious in young subjects than in old, chloroform much more so than ether.

This was representative of many views in 1848.

While in the states Morton and Jackson were fighting over credit for the discovery of anesthesia, which had been suggested long before by Sir Humphrey Davy, and Horace Wells was hospitalized and dying, Simpson was in England defending anesthesia against its medical and religious opponents.

In Simpson's *Answer to the Religious Objections Advanced Against the Employment of Anaesthetic Agents in Midwifery and Surgery,*[7] he states many passages from the Bible as arguments against those religious opponents who felt women should bring forth children in pain and sorrow as the Bible says. His best argument was a quote from Genesis ii. 21, "And the Lord God caused a deep sleep to fall upon Adam; and he slept; and He took one of his ribs and closed up the flesh instead thereof." In 1853, when Queen Victoria gave birth to Prince Leopold under the influence of chloroform, Simpson's religious opponents had been defeated.

Mortality in surgery at that time was exceedingly high. For some operations, such as amputations, the mortality rate was usually greater than 50 per cent. It was thought by many doctors that anesthesia would increase that rate. Studies performed, however, showed that the mortality rates declined, sometimes astonishingly. In obstetrics labour times were shortened.

Simpson published many papers before his death in 1870. By that time the toxic effects of chloroform were being discovered. The rightful credit for the discovery of anesthesia goes to Morton and (partially) to Wells. However Sir James Simpson deserves credit for defending anesthesia against its many opponents, for having the courage to spend so much time and effort, not for his own benefit but for the benefit of the patient.

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H. B. LINDSLEY, M.D., *Editor*

The Great Imposter

Munchausen's Syndrome and Other Factitious Disease

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TRADITIONAL disease categories in medicine include infectious, congenital, neoplastic, and similar designations. Seldom is factitious or self-induced disease listed, yet every year numerous patients present with baffling symptoms that ultimately prove to be self-induced. Factitious disorders are characterized by physical or psychological symptoms that are voluntarily produced by the patient. The word "ipsepathogenesis," or self-originating disease has been coined to recognize the patient's active participation in the disease process. Factitious disease may present in one of three forms: total fabrication, such as complaints of acute abdominal pain in the absence of any such pain; self-inflicted injury such as the production of abscesses by injection of saliva into the skin; or an exaggeration or exacerbation of a pre-existing physical condition, such as allowing the injection of penicillin despite known prior anaphylaxis.

Factitious disorders must be distinguished from malingering, in which the patient's goal is clearly recognizable and could even be considered adaptive. The prisoner who pretends renal colic in order to avoid incarceration is an example of the malingerer; in contrast, patients with factitious disease have no apparent goal other than assuming the patient role. Furthermore, a true malingerer rarely injures himself or agrees to undergo diagnostic procedures or surgery. Instead, he merely complains of a nonexistent pain or disease. Patients with factitious disease, on the other hand, are apparently eager to undergo unpleasant procedures and often spontaneously request surgery.

Hysteria differs from a factitious disorder in that the physical symptoms are *not* under voluntary control, although they may lack an organic basis. Symptoms are not feigned, lied about or deliberately produced, as they are in factitious illness.

The ultimate factitious disorder is Munchausen's syndrome, named for the widely traveled Baron von Muchausen, known for his exaggerated tales. Like the baron, the patient with Munchausen's syndrome travels from hospital to hospital inventing elaborate descriptions of his symptoms to convince physicians of his need for treatment.

The Diagnostic and Statistical Manual III (DSM III) criteria for chronic factitious disorder with physical symptoms ("Munchausen's syndrome") are: Plausible presentation of apparently voluntary symptoms to such a degree that there are multiple hospitalizations; and an apparent goal only to assume the patient role and behavior not otherwise comprehensible.

The itinerant sailor known as the "Indiana Cyclone" developed the technique of achieving "multiple hospitalizations" to its height. He feigned hemoptysis by self-bloodletting to shock levels, then aspirated his own blood. At last count he had 178 documented hospitalizations at various hospitals.

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TABLE I
EXAMPLES OF SELF-INDUCED DISEASE

Presenting problem	*Clinical Manifestations and Mechanisms*	*Clues to Diagnosis*
Fever	fever with no clear diagnosis (FUO) (due to thermometer manipulation)	loss of diurnal pattern; T> 41.1C; absence of tachycardia, skin warmth or toxicity; rapid defervescence; simultaneous, observed oral and rectal T; urine T
	fever with toxic source (due to injection of bacteria or toxins)	polymicrobial infection in otherwise healthy person
Hemoptysis/ Hematemesis	aspiration or swallowing of human or animal blood	negative evaluation; blood typing
Hematuria	adding blood to urine; urethral laceration; (''allergy'' to contrast dye)	absence of WBCs, protein, casts on urinalysis; lysed RBCs; traumatized urethra
Hypoglycemia	covert insulin or sulfonylurea use	insulin antibodies in non-diabetic; high plasma insulin (IRI) levels >1000 μU/ml; triad of high IRI, low blood glucose and low C-peptide; urine drug screen
Hyperthyroidism	hyper metabolism due to exogenous thyroid hormone	absence of goiter, ophthalmopathy, or dermopathy; elevated T3, low T4 with Cytomel
Hypokalemia	weakness, hypokalemia due to diuretic, laxative abuse	low serum K+ with urine K+ >30 mEq/l; ⊕ urine thiazide screen
Neurologic disease	pseudoseizure	rarely falls, bites tongue, or is incontinent; arouse by pinching nose closed and covering mouth
	pseudocoma (may be unresponsive to pain)	normal breathing and reflexes; resistance to opening eyelids; EEG desynchronizes to auditory stimuli
Diarrhea	laxative abuse	stool NaOH ⊕ if laxative contains phenolphthalein
Urinary tract infection	recurrent UTI despite anatomically normal tract; addition of contaminants to sample	mixed and changing bacterial flora; negative suprapubic tap; absence of WBCs on urinalysis
Dermatitis	recurrent bizarre, geometric skin lesions; panniculitis due to injection with foreign proteins	geometric appearance; distribution only on areas reachable by hands; occlusive dressing test; skin biopsy shows crystals

Examples of self-induced disease are listed in *Table I*.

Clinical Features

The characteristic clinical presentations have been well described and colorfully named. They include recurrent abdominal pain simulating a surgical abdomen (''laparotamophilia migrans''), hemoptysis or hematemesis due to self-aspiration of blood (''hemorrhagica histrionica''), and well-timed public seizures, coma, or stroke (''neurologica diabolica''). Even so, ''it is now conceded that presenting symptomatology is limited only by the individual's medical knowledge, sophistication, imagination and daring.''[1]

Associated features include:

- Pseudologic fantastic — uncontrollable, pathological lying in a manner intriguing to the listener. For example, one patient consistently maintained he was an undersea diver who worked with Jacques Cousteau.
- Wandering and evidence of extensive prior treatment, with patients reporting treatment at hospitals hundreds of miles apart. Often there are multiple surgical and other scars, cranial burr holes and lack of venous access sites.
- Medical sophistication. Patients often provide textbook histories of the onset and course of their illnesses, showing a disturbing familiarity with normally unusual diagnostic procedures.

- Disruptive hospitalization. Munchausen's patients tend to be demanding, flaunt hospital rules, and create havoc on the wards. The great majority of hospitalizations end with the patient suddenly leaving against medical advice (AMA).
- Demands for medication. Patients frequently demand analgesics such as meperidine and morphine in high doses, claiming allergy to less potent medications.
- Absence of visitors. Patients typically have no visitors at all, despite prolonged hospitalizations and multiple surgical procedures.

Patient Characteristics

The classic patient is a male in his mid-thirties with marked antisocial behavior and evidence of wandering great distances between hospitals. However, recently an entirely new group of patients has been recognized — socially conformist young women who are nearly always in the medical profession, most commonly in nursing. They wander between local hospitals and tend to have more stable home situations. The complaints of these patients show more medical sophistication — as for example, the young medical student who faked a trophoblastic mole. Traditionally only the older male patients would be described as having Munchausen's syndrome, although both types fit the DSM III criteria of "chronic factitious disorder with physical symptoms." It is important to realize that there is a *spectrum* of illness, of which Munchausen's syndrome is only one extreme.

The underlying psychopathology has not been defined; however, much recent work suggests that these patients often have "borderline" personalities or marginally compensated function with occasional psychotic breaks. The fact that psychotic breaks occur in this illness attests to significant psychopathology.

Clinical Manifestations

Factitious Fever ("hyperpyrexia figmentatic"). While the spectrum of presenting complaints is limited only by the patient's imagination, one of the more common manifestations is factitious fever. This was first described by Petersdorf in 1956, who noted factitious fever in 14 patients, five of whom were nurses. The most common method of producing fever was switching thermometers, substituting one fixed at a higher temperature. Other commonly used methods included holding the thermometer next to a heat source, and manipulating the teeth, gums, or rectum.

Diagnostic clues include: failure of the temperature curve to follow the normal diurnal variation; fever above 41C; absence of tachycardia, toxicity, or skin warmth; and apparent rapid defervescence when the patient's temperature is rechecked under observation. Methods for diagnosing factitious fever include taking simultaneous oral and rectal temperatures, preferably with both thermometers held in place; taking the temperature only under constant observation; repeating all elevated temperatures within 20 minutes for confirmation; and taking freshly voided urine temperature, which should be within 1C degree of oral temperature.

Self-innoculation with various pyogenic substances is another method of producing fever. In a study by the National Institutes of Health,[2] 13 of 19 patients produced fever by injecting themselves with such substances as tetanus toxoid, urine or feces, pure microbial cultures, or foreign proteins (milk). Several otherwise healthy patients in this series had bouts of polymicrobial sepsis which served as a clue to diagnosis, since infections with more than one organism rarely occur in normal patients. Telltale mouth flora were detected in another case of fever due to subcutaneous injection of saliva.

Factitious Hematologic Disorders ("hemorraghic histrionica"). Factitious hemoptysis and hematemesis are often produced by swallowing or aspirating the patient's own blood or that of an animal. Schechter[3] reported the case of an electrician who gave a history of tuberculosis, chest pain, and severe hemoptysis. After a fruitless thoracotomy, the hemoptysis proved to be the result of deliberately aspirated chicken blood.

Factitious Endocrinopathies. A common and difficult clinical problem is that of factitious hypoglycemia, which can be extremely difficult to differentiate from an insulinoma. Factitious insulin use in the non-diabetic can be detected by the presence of insulin antibodies, which occur only with exogenous insulin use. The very high plasma immunoreactive insulin (IRI) levels in these patients (>1000 μU/ml) are another useful discrimination point, since insulinomas rarely generate a value greater than 200 μU/ml. The level of C-peptide, which is secreted from the pancreas in equimolar amounts with endogenous insulin, is also helpful in detecting factitious or excessive insulin use. The triad of hypoglycemia, high IRI, and low C-peptide is highly indicative of exogenous insulin administration.

Sulfonylureas also can induce a prolonged severe hypoglycemia, during which serum IRI will be high — a finding suggestive of insulinoma. However, the possibility of insulinoma can be readily eliminated by a serum assay for sulfonylureas.

Another common factitious disease is exogenous hyperthyroidism (''thyrotoxicosis factitia'') caused by surreptitious self-medication with thyroid preparations. The presentation is one of hypermetabolism without goiter, ophthalmopathy, or dermopathy. All drugs except synthetic triiodothyronine (Cytomel) will increase thyroxine (T4) and triiodothyronine (T3); Cytomel will suppress T4 but elevate T3. The main differential diagnostic consideration is ''silent'' thyroiditis. In both instances, the thyroid scan will show low uptake; however, in ''silent'' thyroiditis, the sedimentation rate is elevated and antithyroid antibodies may be present. Also, serum thyroglobulin will be low in factitious hyperthyroidism.

Factitious Electrolyte Abnormalities. Hypokalemia, produced by laxative or diuretic ingestion, is a fairly common problem. Rosenblum[4] described a 28-year-old automotive industry executive whose clinical presentation was indistinguishable from Bartter's syndrome, including hypertrophy of the juxtaglomerular apparatus. The diagnosis was made when a urine thiazide screen proved positive. Urinary potassium and thiazide screens may be falsely normal if the patient stops the diuretic on the day the urine is collected. To avoid this error, serum and urine potassium should be measured on three successive days; any urine with a potassium level greater than 30 mEq/l should be tested for diuretics.

Factitious hypercalcemia from vitamin D or $CaCo_3$ tablets (Os-Cal) has been reported. Frame[5] described a nurse who ingested 20-30 Os-Cal tablets daily and presented with a serum calcium of 20.6 mg/dl. Elevated serum phosphate and urinary calcium, normal urinary cAMP, and normal serum 25-hydroxycholecalciferol are useful differential points in such cases. Serum parathyroid hormone determinations may be helpful but are notoriously variable; in this patient they were only mildly elevated.

Factitious Neurologic Disease. ''Neurologica diabolica'' typically presents with paroxysmal headaches, loss of consciousness, and pseudoseizures. In pseudoseizures the patient rarely injures himself, falls, bites his tongue, or is incontinent. The patient may be aroused from an apparent postictal state by pinching the nose shut and covering the mouth to prevent breathing.

Feigned coma may be initially difficult to distinguish from real coma, since the patient may be completely unresponsive to painful stimuli; however, normal breathing continues and pupillary, corneal, tendon, and plantar reflexes are preserved. There may be spontaneous flickering of the eyelids, with resistance to attempts to open the eyes. Although highly unlikely, a normal EEG does not completely exclude an organic cause for coma; desynchronization in response to auditory or visual stimuli can verify normal cortical functioning.

Another common presentation is the unilaterally fixed, dilated pupil produced by anticholinergic or sympathomimetic eye drops. A useful diagnostic point is the absence of cranial nerve III paralysis, as well as the ineffectiveness of pilocarpine in constricting the dilated pupil. (In organic lesions, pilocarpine will produce pupillary constriction).

Factitious Gastrointestinal, Genitourinary and Cardiac Disease. Factitious diarrhea is an easily produced variety of factitious disease, usually caused by the surreptitious ingestion of laxatives. If the laxative contains phenolphthalein, a stool NaOH test will be positive. Other laxatives are more difficult to detect.

Factitious hematuria and urinary tract infections are particularly common presentations of factitious illness. Infection and hematuria are typically produced by such methods as forcing a pencil up the urethra, direct instillation of blood or contaminated material into the bladder, deliberate self-catheterization with contaminated catheters, and addition of stale urine to ''fresh'' urine samples. Clues to the diagnosis include very heavy bacterial growth, mixed and changing flora in patients with anatomically normal urinary tracts, ''freshly voided'' urine that contains lysed RBCs or is at room temperature; and absence of other abnormalities, such as WBCs, on urinalysis. Hematuria and bacteriuria may alternate rapidly with clear urine, suggesting that blood and contaminants are being added to the sample. Failure to confirm an infection on suprapubic tap is another clue.

Nephrolithiasis is also frequently feigned, often with the patient claiming allergy to contrast dye to avoid quick diagnosis.

Ischemic cardiac pain is a particularly easy symptom to simulate. Usually such patients have a baseline abnormal EKG which, along with the classic story they invariably give, results in repeated admissions to coronary care units.

Factitious Dermatitis (''dermatitis autogenica''). Factitious skin lesions often have a bizarre appearance and do not fit the picture of any known disease process; they simply look ''phony.'' Most have regular borders, appear discrete and ''punched out,'' or take geometric shapes and criss-cross in a ''grid-iron'' effect. Factitious panniculitis is also fairly common. The nodules or sinuses result from sub-

cutaneous self-injection of various substances, such as petroleum jelly, feces, milk, saliva, vitamin E oil, and various drugs.

Clues to the diagnosis of factitious dermatitis include the odd appearance and distribution of the lesions, which are usually on parts of the body that can be reached by the hands and especially on the side opposite the dominant hand. The skin may be tested with litmus paper if acid or alkali use is suspected. A skin biopsy may reveal crystals. Often encasing the lesions in an occlusive dressing or plaster cast will reveal their factitious nature.

Treatment

Psychiatric treatment should be attempted, although it is usually rejected by the patient. It is probably best not to directly confront the patient, since s/he will nearly always then leave AMA. Instead, the subject should be approached obliquely: "Your chest pain does not appear to be coming from your heart," then leading into a discussion of other areas of concern. This is best accomplished by a team consisting of the medical staff, the psychiatrist, and the social worker. Perhaps the most important role of psychiatric consultation is to help the medical staff, who usually react to the patient's attempted manipulations with a mixture of disgust and anger. In turn, the most important treatment the medical team can offer the patient with factitious illness is protection from himself — the avoidance of potentially dangerous diagnostic and operative procedures.

Self-Assessment Questions

1. Psychosocial characteristics of patients with factitious disease include all of the following except:
 a. Employment in the medical profession
 b. Occasional psychotic breaks
 c. "Doctor shopping"
 d. Hysterical personality disorder
 e. Eagerness to undergo diagnostic procedures
2. The primary difference between malingering and a factitious disorder is:
 a. Factitious diseases tend to be medically more severe.
 b. Malingerers are generally less psychiatrically ill.
 c. Factitious disease is more likely to present as a mythical pain.
 d. Malingerers have a clearly recognizable goal.
3. The finding most suggestive of factitious fever is:
 a. Three positive blood cultures with Staph aureus
 b. Three positive blood cultures with ∝ strep and diptheroides
 c. Urine temperature one degree less than oral temperature
 d. Oral temperature 40.6C with a pulse of 120
4. Factitious hypoglycemia in an insulin-dependent diabetic can be detected by measuring:
 (More than one answer may be correct)
 a. Insulin antibodies
 b. Serum immunoreactive insulin levels
 c. C-peptide
 d. Serum sulfonylurea screen
5. Factitious hypokalemia:
 a. May be distinguished from Bartter's syndrome by absence of hypertrophy of juxtaglomerular apparatus.
 b. Urine thiazide screens are predictably positive if the hypokalemia is due to diuretics.
 c. Urinary potassium is usually high if the hypokalemia is due to diuretics.
 d. May be detected by measuring urinary chloride on three successive days.

(Answers on page 161)

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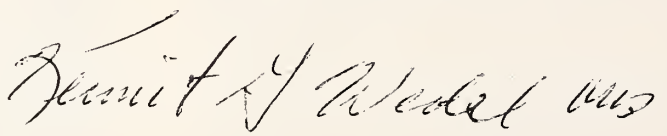
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A Special Thanks to KSM and UKSM . . .

I would like to take this opportunity to thank the members of the KMS-UKSM Liaison Committee. During the past year, this committee has worked toward achievement of common goals and purposes of the Kansas Medical Society and the University of Kansas School of Medicine. They have addressed issues of improved communication, more complete referral information, and increased medical school involvement in KMS activities.

Representatives of UKSM attended Council District meetings with me to respond to concerns and to enhance the relationship with practicing physicians throughout the state.

I thank you for these efforts and encourage continued cooperation and support of both organizations.



President



Drug INFORMATION

JOYCE GENERALI, R.Ph., *Editor*

The Drug Information Service

The Drug Information series, a regular feature of the *Journal,* is provided by the Drug Information Service, Department of Pharmacy, University of Kansas Medical Center. Following are brief answers to questions frequently asked about the Drug Information Service.

What is the purpose of the Drug Information Service?

The purpose of the Drug Information Service is to promote rational and safe drug therapy by improving the availability and use of drug information. The Drug Information Service maintains a collection of medical journals, texts, and other reference sources designed to answer questions about drugs. The Service is staffed by pharmacists experienced in providing such service. These pharmacists are able to selectively retrieve, evaluate and communicate accurate, current, and unbiased information to respond to a specific drug question.

Who can use the Drug Information Service?

It is impossible for most health care practitioners to keep up with the vast amount of published literature concerning drug therapy. Therefore the Drug Information Service functions as a central reference source serving any health care practitioner involved in the treatment of patients. Last year, more than 85 per cent of nearly 1,700 information requests received by the Service originated from physicians, pharmacists, or nurses. Although these practitioners are the primary users of our service, requests from other health care personnel are welcome.

What types of questions can be handled?

The Drug Information Service will provide information concerning any drug problem. In 1982, more than 80 per cent of drug information requests received by the Service involved adverse drug reactions, drug therapy of choice, appropriate drug doseage, drug identification, pharmacokinetics, or drug interactions. Other requests handled by the Drug Information Service include drug-laboratory test interference, indications and contraindications, investigational drug information, intravenous compatabilities, and mechanisms of drug actions.

How will my questions be answered and what is the cost of the service?

Your question will be answered by telephone and/or mail, usually within 1 to 24 hours, depending on the nature of the question and need of the caller. The answer may be a single fact, a summary of an article, or a brief review — all of which will be completely referenced.

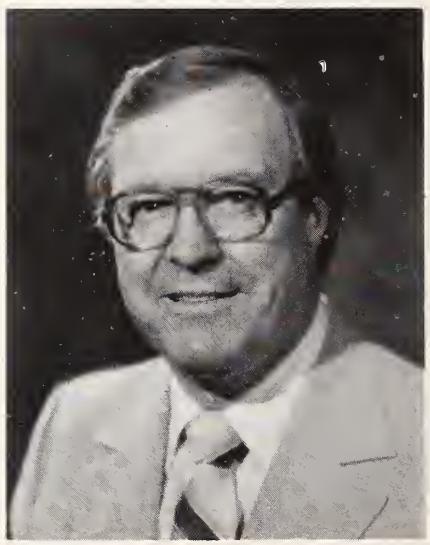
The service is provided free of charge.

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UKSM-KC
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Phone: 913-588-2328

The President's Message



Dear Colleague:

I want to take this opportunity to thank you for your contributions to your Society this past year. It is not possible to name each member individually:

To the Executive Committee, for the time spent in meetings, for your recognition of important issues and guidance, I thank you.

To the Council, for your commitment and wisdom in making policy decision as the true governing body of our Association, I thank you.

To the Committee Chairmen, who dealt with controversial and timely issues, and to all of the members of these committees, I thank you. Without this involvement in your Society, the issues that concern the membership would not be addressed.

To our staff in Topeka, to whom much credit must be given for the day-by-day activities —

Steve: For an excellent job of organizing various functions, for your ability to keep me informed, for your innovative ideas, and your commitment to the success of our society, I thank you;

Val: For your efforts in addressing a personal concern of mine, *i.e.*, providing the opportunity for our Society to become more active in confronting the drug problem in our state, I thank you;

Gary: For your effort, expertise, and guidance of individual committee activity, so essential to reaching our goals, I thank you; and

To the rest of the office staff, for your service to our members; to *Jerry,* for your efforts in the Legislature; and to *Wayne,* for handling our legal affairs; I thank you. Presidents come and go on an annual basis, but your efforts and commitments are paramount for the year-by-year effectiveness of our Association.

To the Council Districts, for your invitations and allowing me to share with you our problems and concerns, and the opportunity to meet your members, I thank you.

Finally, to the membership in general, for your support, for your involvement, and for your tolerance of issues not resolved, I thank you.

Hopefully, we resolved a few issues, looked at many, and kept the Society in a continuum to be prepared for future issues in the year to come.

Dennis G. Wedel MD

President



Editorial COMMENT

This Way Out

Mankind, for all its long experience with the matter, has never quite decided what to make of life. Are we spending an allotted time here between an unremembered past and an unknown future? A sort of existential internship to prepare for what is to come? Is there a larger purpose or is our existence only a time-killing mechanism, serving out our physiological sentence in which we must make our own purpose by trying to improve things (as we see improvement) for ourselves and, incidentally, those who come after? Do the activities and accomplishments we pursue and to which we attach so much importance have any other meaning than to occupy us while we are in residence?

There are only two certainties: we arrive through no choice or action of our own and our eventual departure is assured although we don't know the time or method. The first is long past before we realize it and consequently woven into our intellectual fabric without apparent change in the design. The latter — our own at least — is realized gradually but is displaced into the future so as to have little actual meaning. The occasional experience of the death of those around us rarely gets through the protective personal coat of denial until the threat of age or illness forces a confrontation. One of the most enduring manifestations of this is that the taking of one's own life is relegated to the aberrant. To some, it is one of the most grievous of sins, to others the irrational act of an insane mind, and to others at least a violation of good taste. But to some through the ages, it has been a rational solution to a situation of purely personal character and therefore not to be made a social issue. (Petronius would be pleased.)

Being born is probably the last experience in which an individual will have a totally passive role until death when he is again a victim of circumstances, however much his prior behavior may have contributed to the event. Usually the fact precedes the wish; occasionally, especially in old age or prolonged debilitation, the wish precedes the fact. Rarely do they coincide and man moves through his allotted time, a casual eye on the actuarial tables and intermittent reports of life expectancy — and the annoying persistence of life insurance premiums.

As numerous theories and philosophies have developed to explain the incongruities and inconsistencies — even benefits — of life, the same has been true for death. Life is the one experience with which mankind has some tangible experience, and many consider it abhorrent to think it simply terminates in death, even when life has been far from easy. In general, the religious philosophies assure us that (for those qualified) a preferable and permanent life awaits (although some claim that one may require several tries, thanks to reincarnation, to reach that ineffable state).

It is an interesting if contradictory commentary on life — or is it death? — that despite this not infrequent assurance of something better after death, there is nothing more tenacious in the human character than the determination to hang onto life and the fear that if one actively seeks one's own death, the act will in itself nullify the release and joy so irreversibly sought. Above all, the active, voluntary termination of life known as suicide is to be shunned. If an attempt is made, this act, which epitomizes the most personal of decisions, becomes an occasion for general intervention — for society, the police will intervene; for the body, the medical profession will turn on its full restorative power; for the soul, the church will bring its moral pressures to bear. Their common goal is to retain the individual in this life (and most of them consider it worth the effort) even if the result is to deny him something he desires and for which, in his own mind, a reasonable case can be made. Each of these agents functions in the firm belief that its efforts are the fullest measure of service to the individual, to society, and, in particular, its own group. Failure to act leads to an undercurrent of unease from each of them — as though, if society doesn't act, there will be an

epidemic of the "crime," if the physician doesn't fulfill his obligation to the body, there will be a spreading of the "disease," and if the church doesn't keep a firm spiritual custodianship, limbo will be filled with wandering souls. On a more apparent level, the successful thwarting of the act avoids, for all of them, a large sense of failure and guilt. Meanwhile, although there is currently loud support for individual rights, the majority do not consider the forsaking of life to be one of them.

In spite of these cultural deterrents, there comes a time in the lives of some when life *is* intolerable and the desire to live is replaced by a desire not to live, whatever the unknowns. It really is better to "fly to others we know not of" rather than continue in the current "weary life." Whether this indicates a state of social, medical or spiritual pathology, we have no expectation of introducing at this point any interpretation of such profundity as to make appreciable impact on the general attitudes toward it. Rather, we are interested in certain changes that are occurring in all of these areas and that may reasonably be expected to have an effect in the long run.

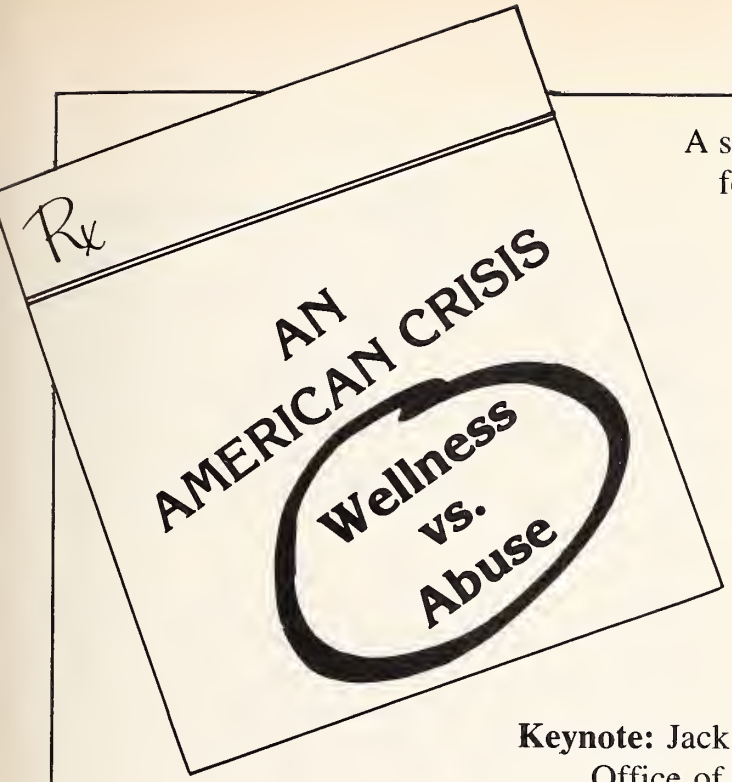
The most obvious, of course, is the increase in life expectancy, itself a product of multiple changes in our lives. This might be divided rather arbitrarily into the natural and artificial (read technologic), but the effect is the same: many people are surviving longer with an extended period of deterioration (leading to the uncomfortable question, how much is enough?), while others are sustained by artificial means continued only because of the obvious reluctance of the attendants to commit any overt act to which death could be traced (again, how much is enough?). In the latter case, the judicial blessing may be bestowed on behalf of society and in the popular phrase of the day, the plug is pulled, and everyone feels better — including, we can assume, the patient. (This assumption is vigorously denied by those who feel otherwise but is *their* assumption any more valid?) This leaves a considerable number, however, for whom there is no plug to pull so they — and we — continue in the "natural" course.

These considerations relate, of course, to the matter of euthanasia, suicide's slightly more mentionable cousin and a subject that has received increasing attention of late. The problem of exploring suicide and euthanasia too far, however, is that it opens doors into some of our more cherished activities in which we consider the benefits sufficient to assume a cost in self-destruction to be entirely reasonable. The medical profession at this point might well feel bemused about its role in conserving life. Society, despite its insistence on the sanctity of that condition and declared dedication to its support, denies the fact in numerous ways. Warfare, a form of self-destruction on the international level, is seen, if properly orchestrated by its interpreters, as one of the highest expressions of national virtue and its successful participants are decorated for valor. We tolerate destruction not only of self but of others on the highways rather than accept (or even agree on) the restrictions that would avoid it. Modern gladiators are lauded and lavishly rewarded for their ability to give and withstand physical abuse — until one dies and then there is a loud demand for reform (which subsides shortly). Those habits of personal conduct that demonstrably display a harmful cumulative effect are controlled only by personal effort — social (especially legal) prohibitions have historically failed. Meantime, the guardians of public safety who are charged with risking their lives to protect ours receive remuneration that implies that neither is worth much in the current coin.

But admittedly those expressions of a confused social attitude are only tangential to the medical function. For physicians, the situation has produced one of its more uncomfortable confrontations. From the dim beginnings of ethics and morality, the medical purpose has been controlled by the precept that physicians must do only what is intended to sustain life and shun that which has even the appearance of assisting death. But the precept has in itself been sustained largely through the limitations of the profession to control such events: physicians did what they could without overt harm and then left the matter to some higher — or more natural — authority, all with the thanks of the social and spiritual arbiters.

What now that increasing numbers are surviving by artificial means or — for the aged — the protective barriers of social and chemical isolation so that they do not succumb to the "natural" causes of death which served so conveniently and acceptably for centuries? In a few instances, as mentioned, the decision has been taken out of medical hands by society in the form of the judiciary. But any relief the medical profession feels by this transfer should be tempered by the realization that it has surrendered a basically medical decision to others (a situation that has recurred with increasing frequency of late, it would seem). This is not to say it is right or wrong — it is clearly impossible today to consider only the medical position — but we do submit it will produce a change in that position — its moral philosophies as well as its pragmatic applications — and that ultimately the process is moving toward some concept

(Continued on page 166)



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Women Physicians

The Career/Family Dilemma

As more women enter medical schools and become physicians, many will confront the dilemma posed by a desire for marriage and children as well as for a successful career. A recent article published in the *Journal of the American Medical Association (JAMA)* offers perspectives from a male and a female physician on the difficult choices women physicians must make.

Women's most significant battle — the battle for equal opportunity to enter medical school — has been won, according to E. Grey Dimond, M.D., Distinguished Professor of Medicine, University of Missouri-Kansas City School of Medicine. He points out that women's representation in medical schools has increased from 6 to 31 per cent during the past decade and that the "essentially all-male profession as we now experience it is well on its way to a balance in numbers between men and women." Women's numbers are also increasing in specialty residencies and academic appointments, he adds.

The effect of this growing number of women physicians on medical practice in the future is unknown, but one thing is certain: "Women physicians will have babies and therefore will have pregnancy leaves from their practices." Dimond suggests that this be construed as a variable similar to those affecting men's careers — military service, greater susceptibility to heart attacks, and earlier death.

Without discounting the impact of marriage and motherhood on a woman physician's career, Dimond points out that choices still must be made. "I hope these new career physicians really are career physicians. It is all going to be wrong if a third of the openings in medical school have been awarded to part-time professionals or misplaced housewives. One hopes the new woman physician learns quickly the value of her considerable profession and its consequent considerable income, and frees herself of many things that can be done well by others."

Dimond believes that women physicians have the obligation to free themselves from domestic chores. And although they can't delegate childbirth, women physicians should consider professional day care or the services of grandparents to help them with child rearing.

Recognizing that relinquishing some of these traditional roles of a wife and mother will be difficult for many women — and for their families — Dimond contends that "having fought for and gained access to the special privilege of being a physician, one can't have it both ways." Still, those women physicians who wish to continue performing some domestic activities should not be criticized as "part-time" physicians, Dimond says. Women will have many years of professional life after their children are grown.

Marilyn Heins, M.D., Associate Dean for Academic Affairs, University of Arizona Health Sciences Center, Tucson, agrees with Dimond's assessment of women's progress in medical education. "The sheer numbers of women make it possible for women not only to *be* in medical school, but to feel comfortable there. Women are beginning to be accepted even in those residency programs once thought to be for men only," Heins says.

Combining marriage and child rearing has been a personal challenge for Heins, who married immediately after her residency and has raised four children. "The parenting *vs* career dilemma has three major components," Heins explains. "First, there is the biologic imperative. The childbearing years are finite and cannot be extended. . . . There is also the psychological imperative. Everyone wants to be a good parent. Everyone knows how important optimal parenting is to the well-being of the child and future adult. Perhaps physicians (knowing a good deal about what can go wrong) want to be even better parents than anyone else, which may lead to guilt and fears about part-time parenting. The sociological imperative is concerned with the roles of men and women as parents. In some families, even a minor deviation from the traditional father and mother roles can cause enormous marital difficulties."

The woman physician must play either a "superwoman" role or work less than full time, according to Heins. And for women who would like to advance in academic medicine, denying time to teaching, research, and service to their institutions — the three areas of performance on which promotion and tenure depend — could slow their careers to the extent that they might never catch up to their male colleagues.

Heins agrees that women physicians should learn to delegate household tasks. But there are problems beyond just having to find someone to cook and clean and care for the children. Tax laws do not

(Continued on page 162)

Obituaries

Glenn R. Peters, M.D., 70, a past KMS president, died February 3, 1983, at his home in Kansas City. A graduate of The University of Kansas School of Medicine in 1937, Dr. Peters practiced surgery in the Kansas City area for nearly 40 years prior to his retirement in 1980. He was the founder of the Glenn R. Peters Museum of Medical History at Providence-St. Margaret Health Center. He served as KMS president in 1959-1960. Memorial contributions may be made to First Pilgrim Congregational Church or to Providence-St. Margaret Health Center.

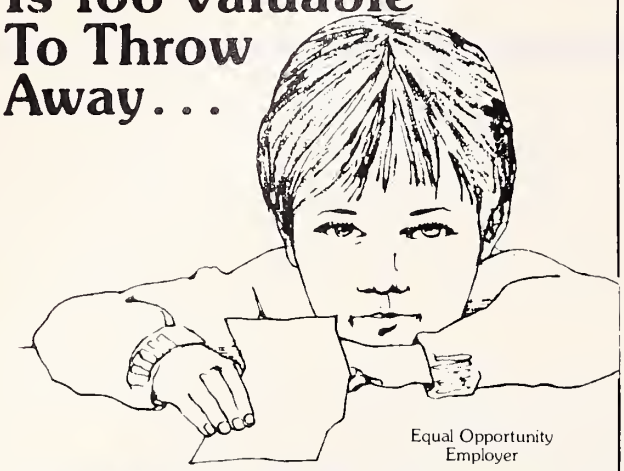
G. Ralph Combs, M.D., 104, Leavenworth, University of Pennsylvania 1902, died January 17, 1983.

Curtis A. Nystrom, M.D., 59, Topeka, The University of Kansas School of Medicine 1954, died February 12, 1983.

Arthur L. Pincomb, M.D., 62, Olathe, The University of Kansas School of Medicine 1951, died January 3, 1983.

Peter K. Wiens, M.D., 58, Ness City, The University of Kansas School of Medicine 1955, died January 9, 1983.

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The Catalogue of Audiovisuals of the Clendening Medical Library is now available from the Educational Resource Center, University of Kansas College of Health Sciences and Hospital, Kansas City, Kansas 66103. The newly produced catalogue lists more than 1,300 audiovisual items available for loan from the Educational Resource Center of the University of Kansas Medical Center. The catalogue contains four separate indices: Title, Subject, Name (KUMC credits), and Series. For additional information, contact Christina Campbell, (913) 588-7343.

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Individualized Treatment

(Continued from page 108)

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The Great Imposter

(Continued from page 151)

Answers

1. d
2. d
3. b
4. b, c, d
5. c

Register for State Meeting by April 13

The Kansas Foundation for Medical Care, Inc. (KFMC), Topeka, is seeking to fill the half-time position of Medical Director of KFMC.

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Postpartum Psychiatric Disorders

(Continued from page 137)

ments in memory or orientation were seen. Typical symptoms were also observed in those patients diagnosed as having postpartum depression. These symptoms include tearfulness, depressed mood and affect, despondency, increased or decreased need for sleep, anxiety, anorexia, disturbances of thought and concentration, but a general lack of homicidal or suicidal ideation.

Although specific conclusions cannot be drawn concerning the etiology of postpartum psychiatric disorders, the current theories in the literature are generally supported by this study. The presence of predisposing conditions has been discussed above. Other factors present in the patients reviewed included evidence of conflict concerning the mothering role as exhibited by a fear of inadequate ability to care for the baby and feelings of hostility. Preexisting marital conflicts and psychiatric problems were also noted.

Recommended therapy for postpartum psychosis includes antipsychotic medication (Haldol, Navane, Thorazine in this study) depending on the specific symptomatology. Hospitalization and separation from the infant is also recommended until the patient is able to handle the stresses and responsibilities of motherhood. Therapy for postpartum depression requires antidepressive medication such as the tricyclic antidepressants. If drug therapy is unsuccessful, electroconvulsive therapy may be necessary, but was not needed for patients in this study.

Our conclusions generally confirm previously reported studies. Predisposing factors, symptom onset following delivery, and general symptomatology of the patients studied showed good correlation with previous reports in the literature. Treatment methods proposed in the literature were successful in patients from this study. Long term followup and prognosis for patients with postpartum psychiatric disorders are poorly defined, but patients with previous psychiatric disease have the poorest prognosis.

A bibliography of suggested reading is available from the authors.

Women Physicians

(Continued from page 158)

permit deduction of the true costs of household help as they do the salary of a medical office nurse, for example. America also trails some other countries, Heins says, in child care arrangements, pregnancy benefits, and maternity and paternity leaves.

Male physicians could help their female colleagues, Heins suggests, by recognizing the inevitability that women physicians will take time to have families; by removing career barriers to pregnant physicians or those with young children; and by acknowledging that a woman physician who advances more slowly in her career because of family responsibilities is not necessarily less intelligent, less committed, or less worthy of promotion than male physicians on faster career tracks.

"The woman who chooses to work two days a week in an emergency room to spend more time with her child is also a physician who can serve her patients with care and compassion," Heins says. Such women who, with their husbands, can cope successfully with the career-family dilemma can serve as role models to the millions of American families in which the mother is employed outside the home, she concludes.

Whether women will continue to advance into the mainstream of American medicine or return permanently, over the long term, to home and hearth is uncertain according to Dimond. But for the foreseeable future, he says, "women will be physicians, and something different is to be expected in American medicine."

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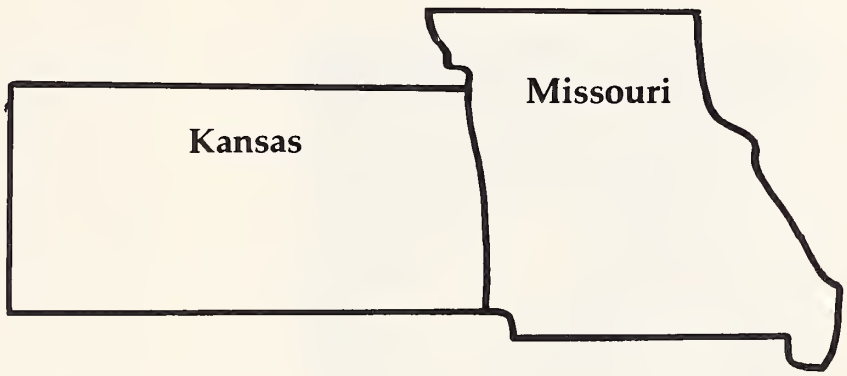
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Nondrug Therapies

Nondrug therapies can benefit all hypertensive patients, particularly those with mildly elevated blood pressure, according to Norman Kaplan, M.D., F.A.C.P., professor of internal medicine at the University of Texas Health Science Center, Dallas. Dr. Kaplan addressed the 1982 American College of Physicians' (ACP) annual scientific meeting.

For patients with mild hypertension — defined as a diastolic pressure between 90 and 104 mm Hg — weight reduction and sodium restriction alone may control blood pressure so that drug therapy will be unnecessary or at least postponed, according to Dr. Kaplan. For patients who do need medication to achieve adequate control, he said, addition of nondrug therapies to the regimen can potentiate the beneficial effects of drugs and ameliorate some of their negative effects.

Some patients may not be willing to change their lifestyles to comply with a nondrug regimen, however, Dr. Kaplan observed. "Although they would rather be given medication and left with their bad habits," he said, "the prudent physician should present the best current evidence and encourage the patient to make changes that are feasible and potentially helpful."

Still, before any therapeutic approach is initiated in mild hypertension, the physician must first determine, through a series of multiple readings during an extended time frame, that the patient actually does have hypertension, Dr. Kaplan said. Similarly repeated measurements also are necessary before the benefit of any type of therapy can be established, he cautioned.

A study conducted in Israel with moderately obese hypertensive patients provided good evidence that weight reduction can be accompanied by a significant fall in blood pressure. Patients lost an average of 9.5 kg during four months, and the average fall in blood pressure was about 30/20 mm Hg. This effect was independent of sodium restriction because urinary sodium content of those in the experimental group equaled that of patients in the control group, he noted.

In view of the frequent failure of dieters to maintain weight loss, however, weight reduction alone is probably not sufficient for longterm blood pressure control in obese hypertensive patients. Dr. Kaplan believes that excessive sodium intake is a likely cause of hypertension and that blood pressure can be significantly reduced by decreasing the usual daily intake of 150-200 mEq to 75-100 mEq. This moderate level of sodium restriction is not hard to achieve if patients are instructed to eat more fresh and frozen foods and avoid canned and other processed foods that almost always have a high sodium content. Eating more fresh foods can also alleviate potassium loss in those who are on diuretic therapy.

Although there is no definitive evidence that regular isotonic or dynamic exercise helps reduce blood pressure, there is no reason to restrict these activities in patients with moderate hypertension. Dr. Kaplan advocated advising patients to adopt a gradual approach to increasing levels of exertion and pointed out that some antihypertensive agents, such as beta blockers, may decrease a patient's exercise capacity. Isometric or static exercise should be ruled out for hypertensive patients, according to Dr. Kaplan. Not only do they provide no lasting benefit, but the rise in pressure that accompanies isometric contractions could precipitate a vascular crisis, he warned.

Few of the various studies of behavioral relaxation techniques have shown a sustained effect on blood pressure reduction, but such therapies, along with daytime naps and adequate nighttime sleep, could have some benefit for hypertensive patients.

Too much or too little alcohol may be harmful for the hypertensive patient, Dr. Kaplan said, citing the greater prevalence of hypertension in both teetotalers and in those whose alcohol intake averages more than 2 oz daily. Moderate intake of 1-2 oz/day, however, should probably be encouraged because it also has the side benefit of raising high density lipoprotein cholesterol, which has a cardioprotective effect.

Dr. Kaplan advised physicians to turn to drug therapy in cases of uncomplicated hypertension only when diastolic pressures remain at or above 100 mm Hg after six months of nondrug therapy. If the diastolic pressure starts below 110 mm Hg and decreases and stays below 100 mm Hg, staying in good shape and restricting sodium may be all that is needed. Patients on nondrug regiments, however, need to be monitored just as closely as those on medication to make sure that the diastolic pressure does not rise above 110 mm Hg, at which point drug therapy is mandatory.

Breast Cancer Screening

(Continued from page 133)

A third area of concern was the cost involved with breast cancer screening due to the high price of diagnostic mammography. During the program, the screening procedure was provided free of charge to participants since the Kansas BCDDP was funded by the NCI and ACS. Since the end of the funding period, the University of Kansas Breast Cancer Detection Center has continued to operate but the steadily rising cost of screening continues to be a major concern of the Center. It is unfortunate that the price of diagnostic mammography is prohibitive when it is such an effective tool in breast cancer detection. Until the time when screening mammography is perceived as being as valuable in detecting breast cancer as the Pap smear is in detecting cervical cancer, the cost of screening will remain a major deterrent to breast cancer control.

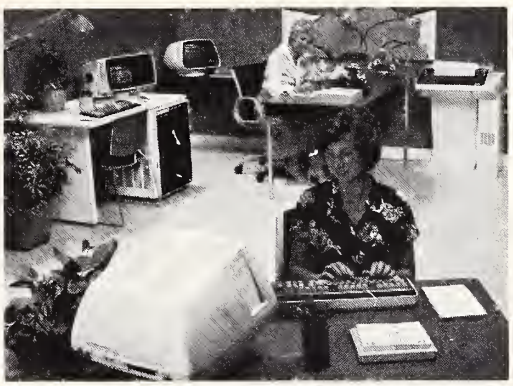
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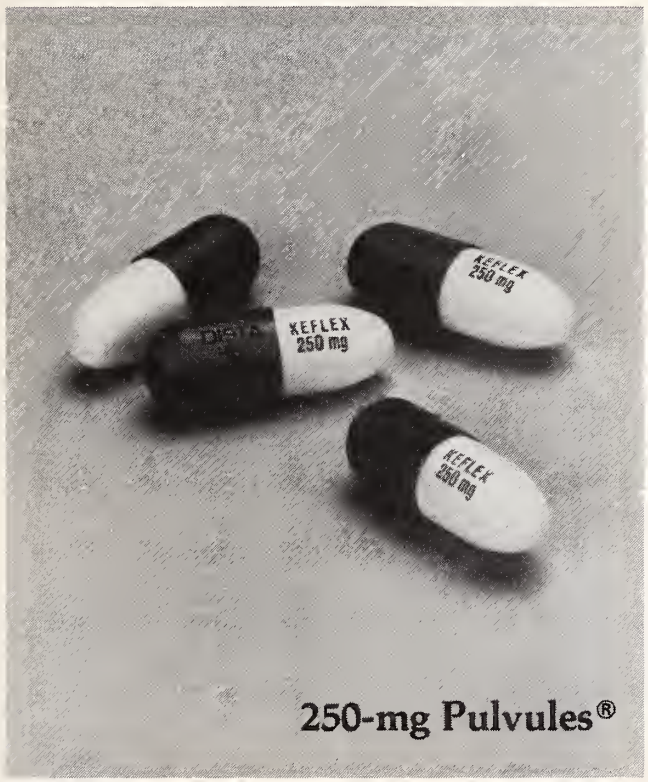
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of euthanasia although semantic manipulations may cloud the recognition of the fact.

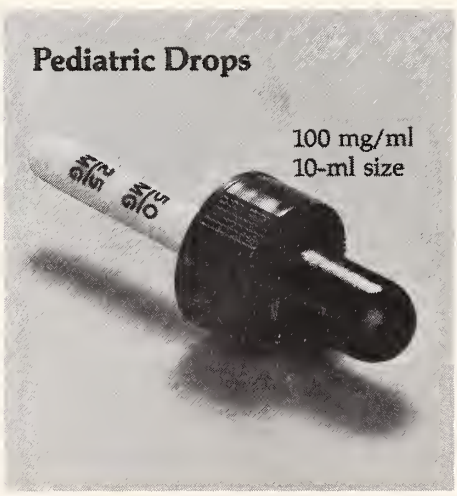
Nor is this to overlook a perhaps more powerful social pressure — at least more imperative in action: the economic impact. It is already apparent that the cost of these changes in marketplace terms affects not only the quantity and character of the changes but the direction and rate. And survival of the aged or technologically supported ill is only one example of potential expressions that are sure to become manifest.

Despite the social and economic dilemmas, it is still all but impossible for the medical mind to say, "This is as far as we go," whether in terms of research or of maintenance of the individual patient. If the fundamental service of the physician is to serve the welfare of the patient, what is the substance of that welfare — maintenance under all circumstances? The medical effort does, in fact, constitute a probing process in which the fundamental purpose is to explain life primarily as a means of extending it. The medical antennae that receive a negative signal from one source will turn to another with slow but continuing progress. And a continuing reevaluation of attitudes toward the voluntary termination of life — active or passive — must be an integral part. — *D.E.G.*

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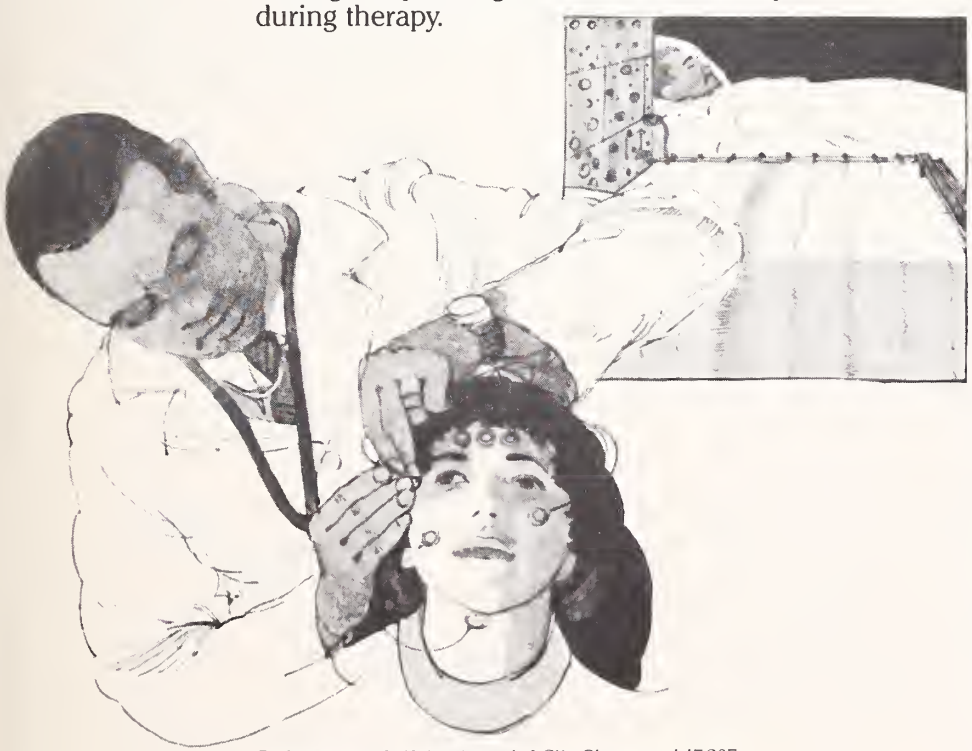
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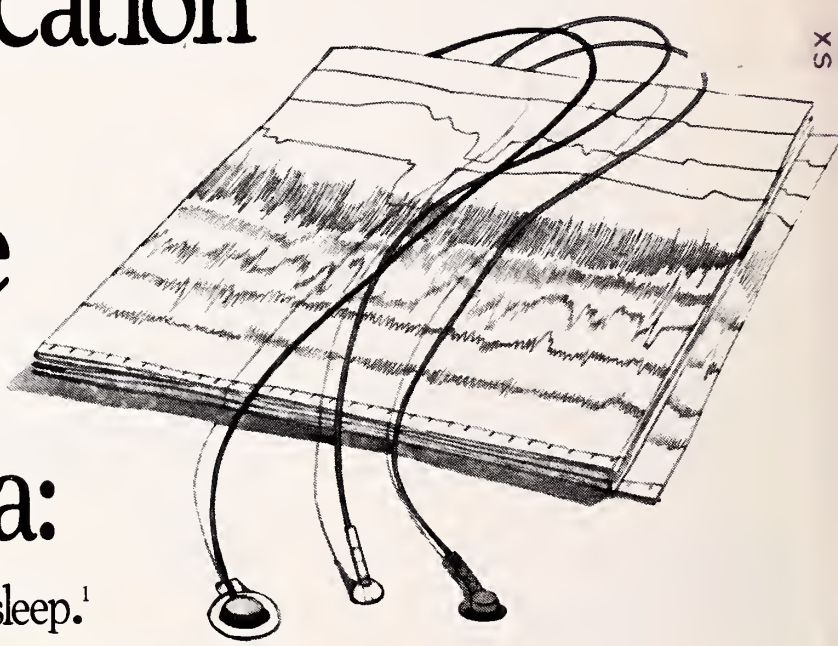
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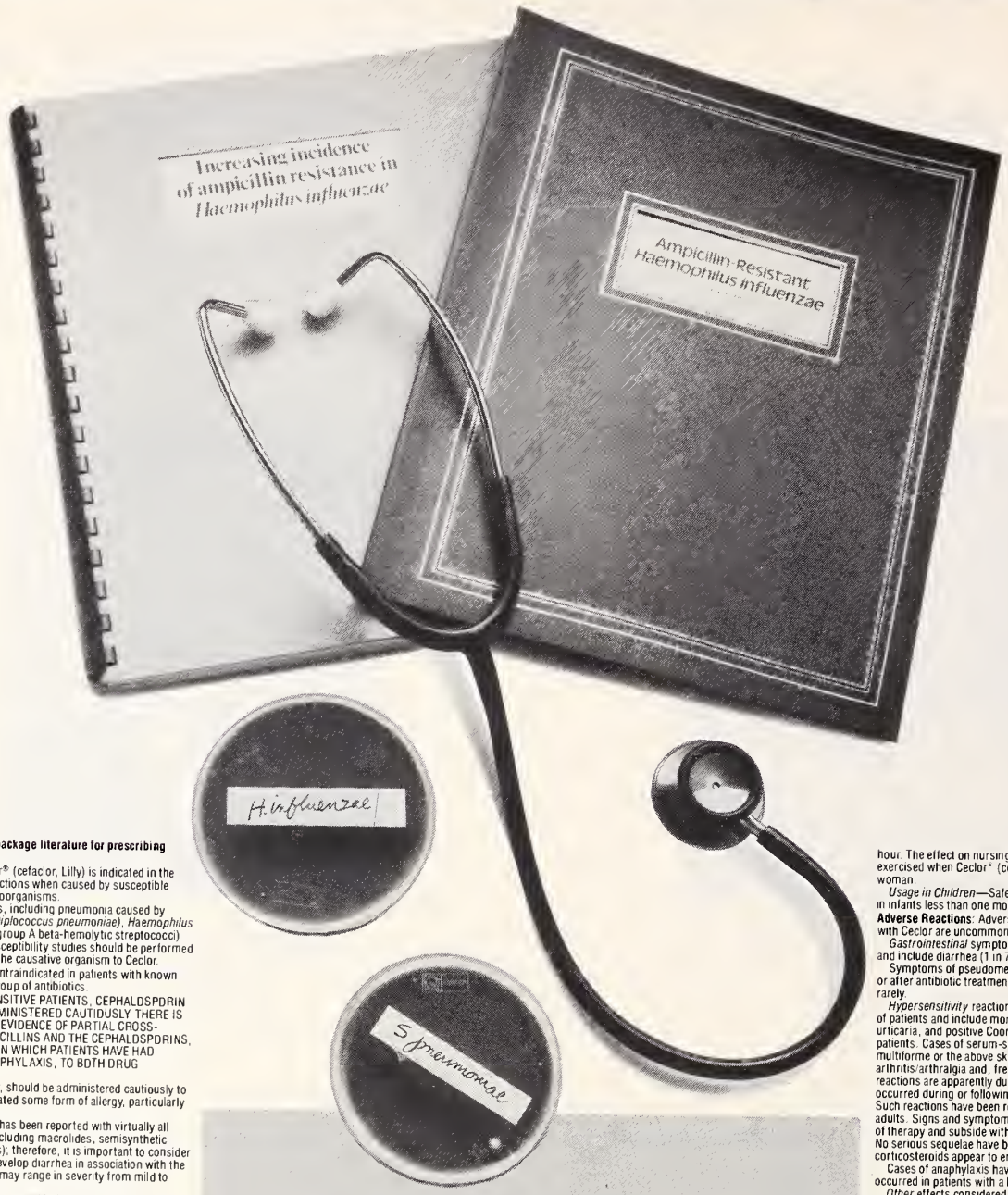
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Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile.* Other causes of colitis should be ruled out.

Precautions: *General Precautions*—If an allergic reaction to Ceclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

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Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers—Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor® (cefaclor, Lilly) is administered to a nursing woman.

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Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

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Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations of SGDT, SGPT, or alkaline phosphatase values (1 in 40).

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[0617B2R]

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae.*[8]
Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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8. Principles and Practice of Infectious Diseases (edited by G.L. Mandell, R.G. Douglas, Jr., and J.E. Bennett), p. 487. New York: John Wiley & Sons, 1979.

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Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

300035

QUICK REFERENCE GUIDE

Thursday, May 5

*	8:00 a.m.	Registration	Holiday Inn-Holidome
*	8:45 a.m.	Sports Day for the Non-Athlete and Athlete	Robinson Gymnasium
	11:00 a.m.	Golf	Alvamar Country Club
†	11:00 a.m.	Golf (18 holes)	Lawrence Country Club
*	2:00 p.m.	Tennis (Doubles and Mixed Doubles)	Alvamar Tennis & Racquet Club
†	2:00 p.m.	Golf (9 holes)	Lawrence Country Club
	2:00 p.m.	Trap Shoot	Cedar Hill Trap Club
*	6:00 p.m.	Social Hour (Cash Bar)	Elks Club
*	7:00 p.m.	Sports Banquet	Elks Club

Friday, May 6

	7:00 a.m.	Kansas Foundation for Medical Care	Brazilian D
	7:30 a.m.	Registration Opens	Lobby
†	8:00 a.m.	Registration, Hospitality and Boutique	
	9:00 a.m.	First House of Delegates and Reference Committee	Brazilian A&B
†	8:45 a.m.	Pre-Convention Board of Directors	Brazilian C
†	10:30 a.m.	Tour of Spencer Art Museum	University of Kansas
	12:30 p.m.	Delegates Luncheon	Brazilian C&D
†	12:30 p.m.	Luncheon Honoring Past Presidents	University of Kansas Union
†	1:30 p.m.	General Session I	University of Kansas Union
	2:00 p.m.	Reference Committee (continued if necessary)	Brazilian A&B
	2:00 p.m.	AMA Tele/Net	
*	4:00 p.m.	KaMPAC Hospitality Suite	Brazilian C
	4:00 p.m.	Kansas Society of Internal Medicine Reception	Regency D11
*	5:30 p.m.	Social Hour (Cash Bar)	Regency A,B,C & D
*	6:30 p.m.	Dinner Sponsored by the Kansas Medical Society Auxiliary Followed by AMA-ERF Auction & Prairie Party	

Saturday, May 7

	7:30 a.m.	Registration Opens	Lobby
	7:30 a.m.	Past Presidents Breakfast	Regency D1
†	7:30 a.m.	Past State Presidents Breakfast	Brazilian D
†	8:00 a.m.	Registration, Hospitality & Boutique	
	8:00 a.m.	Scientific Program - "Cancer in Kansas - 1983"	Brazilian A&B
†	9:00 a.m.	General Session II	Regency C
	11:30 a.m.	Kansas OB-Gyn Society	Regency A
	11:30 a.m.	Kansas Society of Pathology	Regency D11
	11:30 a.m.	Kansas Society of Anesthesiology	Regency B
	11:30 a.m.	Section on Ear, Nose & Throat	Regency D1
†	12:30 p.m.	Luncheon Honoring KMS Advisory Committee and Installation of Officers	Lawrence Country Club
†	2:30 p.m.	Post Convention Board of Directors	Lawrence Country Club
*	5:30 p.m.	Installation of Kansas Medical Society President	Regency A,B,C&D
*	6:30-8:30	Kansas Medical Society Presidents Reception	

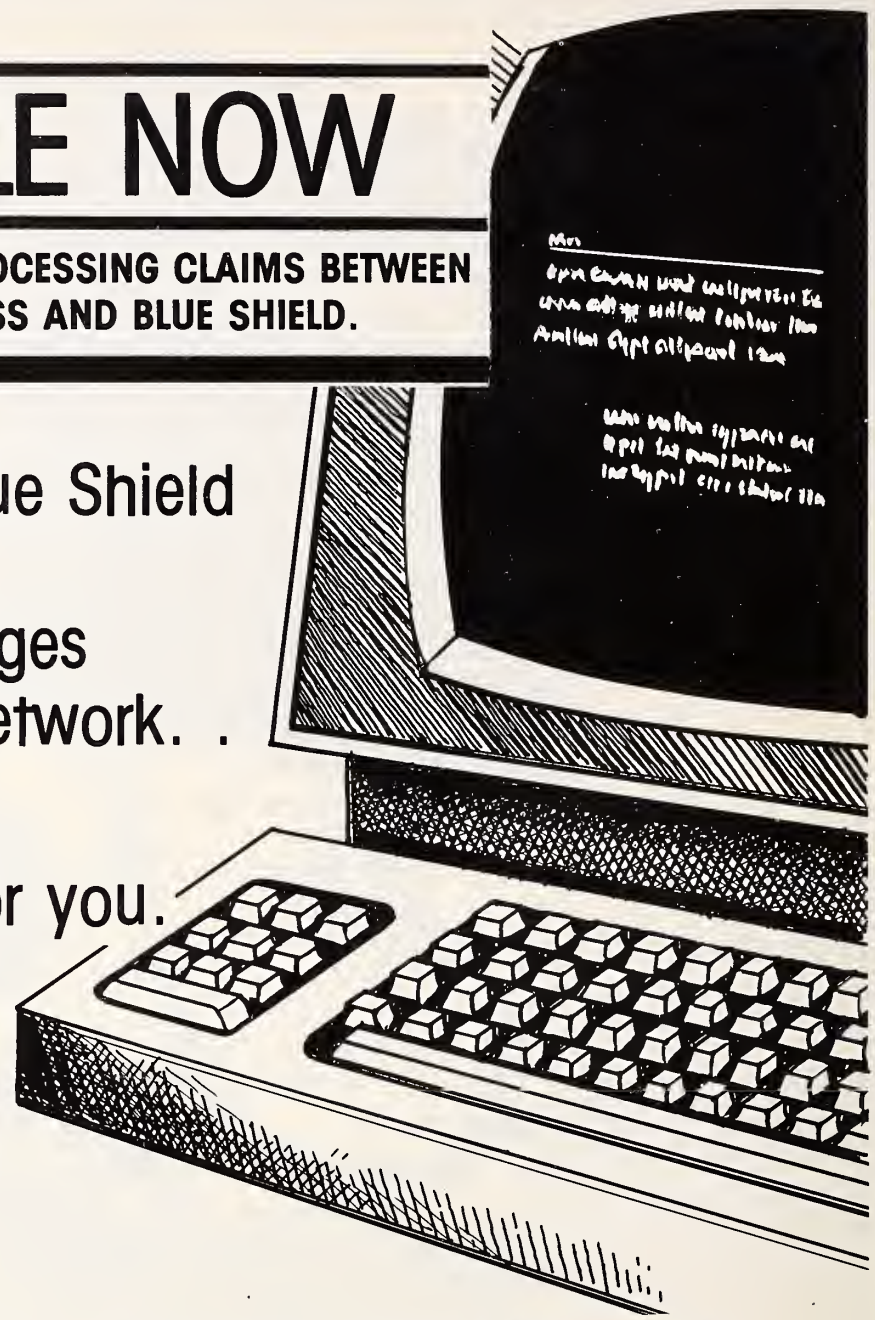
Sunday, May 8

	7:00 a.m.	Registration Opens	Lobby
	7:00 a.m.	Delegates Breakfast	Regency A&B
	8:00 a.m.	Second House of Delegates	Brazilian A&B
	11:00 a.m.	Council	Regency A
	11:00 a.m.	Kansas Urological Society	Regency D1

* **Joint KMS-Auxiliary Function** † **Auxiliary Function**

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Rufen® offers your patient effective relief, both as first therapy or after other potent medications fail. In comparable trials with indomethacin[1,2] sulindac,[3,4] and other antiarthritic agents,[5-7] findings consistently demonstrate high improvement with ibuprofen (Rufen) by such objective and subjective measures as reduction of swelling, improved grip strength,[1] reduced morning stiffness,[1] better ambulation,[1] improved range of motion,[4] reduction and relief of pain.[1-5]

Low Score in Side-Effect Risk

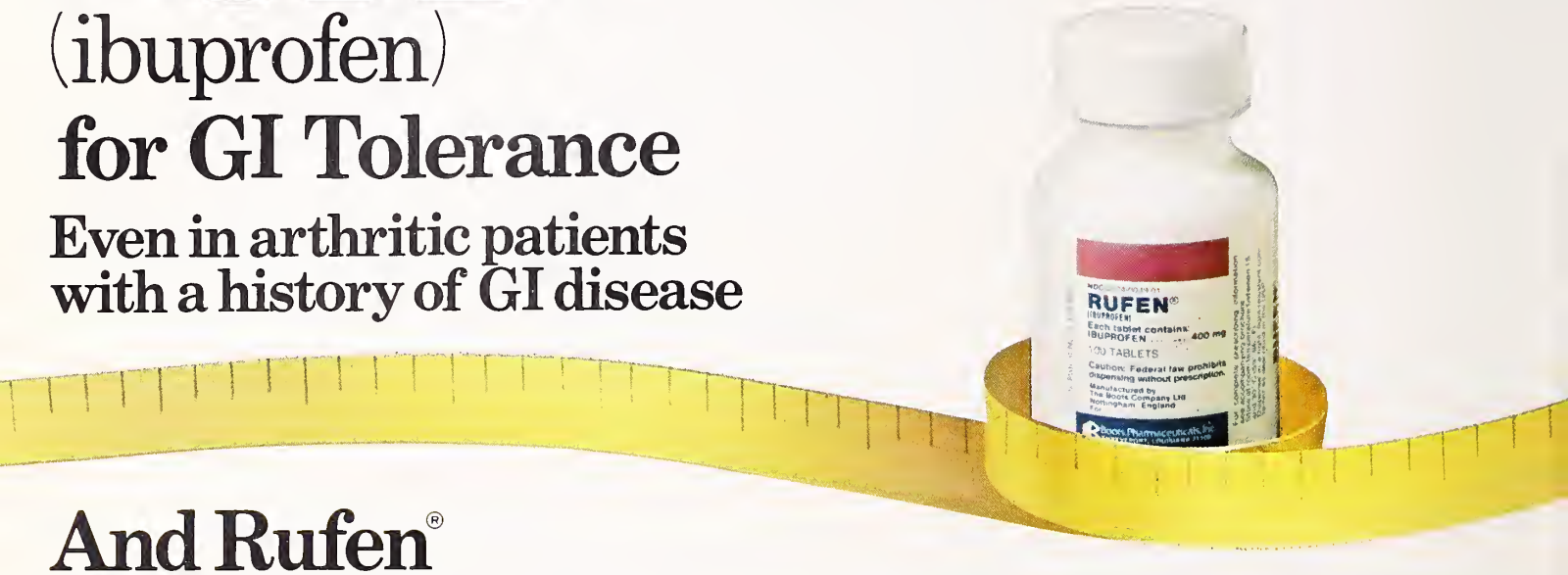
Through more than 13 years of worldwide use, ibuprofen continues to demonstrate exceptional gastrointestinal tolerance vis-a-vis aspirin and other antiarthritic agents. In a recent series of double-blind trials of ibuprofen, naproxen and other NSAID's, only placebo was shown to produce less G.I. lesions than ibuprofen on gastroscopic examination.[5]

Boots Pharmaceuticals, Inc.
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Pioneers in medicine for the family

See next page for brief summary of prescribing information.

Measure RUFEN® (ibuprofen) for GI Tolerance

Even in arthritic patients with a history of GI disease



And Rufen® Measures Up Best

Over a five-year period, ibuprofen was administered to 64 patients with known peptic ulceration and 42 with known gastric intolerance to other antiarthritis drugs.

Twenty-six patients remained in treatment, 23 left treatment following remission, and 35 dropped out for reasons unrelated to side effects. In this specially selected group of GI-intolerant patients, only 13 (12.3%) discontinued ibuprofen because of GI intolerance.

"Any drug used in the control of the symptoms of the chronic arthritis must be tolerated for long periods without undue gastric discomfort...From this study it appears that ibuprofen is eminently suitable."[8]

Peptic ulceration and GI bleeding, sometimes severe, have been reported. Rufen® should be given under close supervision to patients with a history of upper GI tract disease.

References: 1. Royer GL, Jr, Moxley TE, Hearron MS, et al: *J Int Med Res* 3:158-171, 1975. 2. Royer, GL, Jr, Moxley TE, Hearron MS, et al: *Curr Therap Res* 17:234-248, 1975. 3. Brackertz B, Busson M: *Brit J Clin Pract* 32:77-80, 1978. 4. Tausch J, Fasching U: *Brit J Clin Pract: A symposium supplement, IXTH European Congress of Rheumatology*, Wiesbaden, Germany, Sept 2-8, 1979, pp 53-61. 5. Lanza FL, Royer GL, Jr, Nelson RS et al: *Dig Dis & Sci* 24:823-828, 1979. 6. Pavelka K, Susta A, Vojtisek A et al: *Rheumatol and Rehab* 12:68-73, 1973. 7. Tretenhahn W: *Brit J Clin Pract: A symposium supplement, IXTH European Congress of Rheumatology*, Wiesbaden, Germany, Sept 2-8, 1979, pp 45-52. 8. Cardoe N: Curr Med Res & Opinion 3:518-520, 1975.

Boots Pharmaceuticals, Inc.
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RUFEN® (ibuprofen) Tablets

INDICATIONS AND USAGE: Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in the long-term management of these diseases. Safety and effectiveness have not been established for Functional Class IV rheumatoid arthritis.

Relief of mild to moderate pain. Treatment of primary dysmenorrhea.

CONTRAINDICATIONS: Patients hypersensitive to ibuprofen, or with the syndrome of nasal polyps, angio-edema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory drugs (see WARNINGS).

WARNINGS: Anaphylactoid reactions have occurred in patients hypersensitive to aspirin (see CONTRAINDICATIONS). Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Peptic ulceration, perforation, or gastrointestinal bleeding can end fatally, however, an association has not been established. Rufen should be given under close supervision to patients with a history of upper gastrointestinal tract disease, and only after consulting the ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be attempted. If Rufen must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

PRECAUTIONS: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If developed, discontinue Rufen and administer an ophthalmologic examination.

Fluid retention and edema have been associated with Rufen; caution should be used in patients with a history of cardiac decompensation.

Rufen can inhibit platelet aggregation and prolong bleeding time. Use with caution in patients with intrinsic coagulation defects and those taking anticoagulants.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy, this therapy should be tapered slowly when adding Rufen.

DRUG INTERACTION: *Coumarin-type anticoagulants.* The physician should be cautious when administering Rufen to patients on anticoagulants.

Aspirin. Concomitant use may decrease Rufen blood levels.

PREGNANCY AND NURSING MOTHERS: Rufen should not be taken during pregnancy nor by nursing mothers.

ADVERSE REACTIONS: Incidence greater than 1%. **Gastrointestinal:** The most frequent adverse reaction is gastrointestinal (4 to 16%). Includes nausea*, epigastric pain*, heartburn*, diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence). Central Nervous System: dizziness*, headache, nervousness. Dermatologic: rash* (including maculopapular type), pruritus. Special Senses: tinnitus. Metabolic: decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS). *Incidence 3% to 9%.

Incidence less than 1 in 100. Gastrointestinal: gastric or duodenal ulcer with bleeding and/or perforation, hemorrhage, melena. Central Nervous System: depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma. Dermatologic: vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome and alopecia. *Special Senses:* hearing loss, amblyopia (blurred and/or diminished vision, scotomata and/or changes in color vision) [see PRECAUTIONS]. Hematologic: neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs' positive), thrombocytopenia with or without purpura eosinophilia, decreases in hemoglobin and hematocrit. Cardiovascular: congestive heart failure in patients with marginal cardiac function, elevated blood pressure. Allergic: syndrome of abdominal pain, fever, chills, nausea and vomiting, anaphylaxis, bronchospasms (see CONTRAINDICATIONS). Renal: acute renal failure in patients with preexisting significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria. Miscellaneous: dry eyes and mouth, gingival ulcers, rhinitis.

Causal relationship unknown. Gastrointestinal: pancreatitis. Central Nervous System: paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri. Dermatologic: toxic epidermal necrolysis, photoallergic skin reactions. Special Senses: conjunctivitis, diplopia, optic neuritis. Hematologic: bleeding episodes. Allergic: serum sickness, lupus erythematosus syndrome, Henoch-Schonlein vasculitis. Endocrine: gynecomastia, hypoglycemia. Cardiovascular: arrhythmias (sinus tachycardia, bradycardia, and palpitations). Renal: renal papillary necrosis.

OVERDOSAGE: Acute overdosage, the stomach should be emptied. Rufen is acidic and excreted in the urine, alkaline diuresis may benefit.

DOSAGE AND ADMINISTRATION: Rheumatoid arthritis and osteoarthritis, including flareups of chronic disease: Suggested dosage 400 mg t.i.d. or q.i.d.

Dysmenorrhea: 400 mg every 4 hours as necessary.

Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for the relief of pain. *Do not exceed 2,400 mg per day.*

CAUTION: Federal law prohibits dispensing without prescription.

The Kansas Medical Society Auxiliary

58th Annual Convention, The Holiday Inn-Holidome, Lawrence

THURSDAY, MAY 5

8:45 Sports Day Program
University of Kansas Campus
11:15 Food demonstration and lunch provided by
Douglas County Auxiliary
11:00-5:00 Registration and Hospitality
Seco Room, Holidome
6:30 Sports Banquet with KMS
Elks Club

FRIDAY, MAY 6

8:45 Pre-Convention Board Meeting
Brazilian Room C
11:00 Tour of Spencer Museum
University of Kansas
12:30 Luncheon honoring Past State Presidents
University of Kansas Union
1:45 General Assembly — Session I
University of Kansas Union
5:30 Social Hour (Cash Bar)
Regency A, B, C
6:30 Dinner honoring Betty Payne (Torrence), AMAA President; Kermit Wedel, M.D., KMS President; and Joseph Boyle, M.D., AMA Trustee, followed by AMAERF Auction and Prairie Party

SATURDAY, MAY 7

7:30 Past State Presidents' Breakfast
Brazilian Room D
9:00 General Assembly — Session II
12:00 Luncheon honoring KMS Advisory Board and KMS Staff
Installation of Officers
Program: Scandinavian Dancers
Lawrence Country Club
2:30 Post-Convention Board Meeting
Lawrence Country Club
6:00 KMS Installation of Officers and Reception
8:30 Regency A, B, C

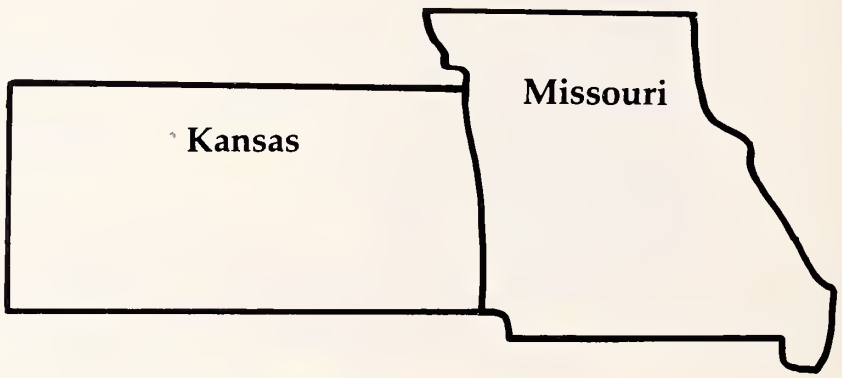
The Boutique and Hospitality Room will remain open throughout the Convention except during special events.

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NOMINATING COMMITTEE

The Nominating Committee of the Kansas Medical Society, consisting of Herman W. Hiesterman, M.D., Chairman; Drs. Herbert Fransen, David M. Pugh, and Kermit G. Wedel, submits to the House of Delegates the following list of nominations for the elective offices of the Society. Wherever more than one nomination appears, they are presented in alphabetical order.

President

Jimmie A. Gleason, M.D., Topeka. Born in 1933. Graduated from the University of Kansas School of Medicine in 1958. Practices Obstetrics and Gynecology. Is now President Elect and has chaired the Legislative Committee for many years.

President Elect

F. Calvin Bigler, M.D., Garden City. Born in 1931. Graduated from Yale University School of Medicine in 1957. Practices General Surgery. Is Past President of the Kansas chapter, American College of Surgeons, and is now serving as First Vice President of KMS.

First Vice President

Clair C. Conard, M.D., Dodge City. Born in 1927. Graduated from the University of Kansas School of Medicine in 1955. Practices Internal Medicine. Now serves as AMA Delegate and has been Speaker of the KMS House of Delegates.

Second Vice President

Franklin G. Bichlmeier, M.D., Kansas City. Born in 1933. Graduated from the University of Kansas School of Medicine in 1958. Practices Cardiovascular Surgery. Is currently serving as Chairman of the Judicial Committee and as Vice Speaker.

David H. Clark, M.D., Salina. Born in 1936. Graduated from the University of Kansas School of Medicine in 1962. Is in Family Practice. Has served on the Continuing Medical Education Committee and is currently a member of the Mediserve Board.

Ivan E. Rhodes, M.D., Wichita. Born in 1925. Graduated from the University of Oklahoma School of Medicine in 1949. Practices Radiology. Has been a Councilor and is currently serving as Chairman of the Impaired Physician Committee.

Constitutional Secretary

K. William Bruner, Jr., M.D., Wichita. Born in 1944. Graduated from Harvard Medical School in 1970. Practices Pathology. Is active in KMS committee work and is currently serving as Constitutional Secretary and as Councilor.

Treasurer

Richard Meidinger, M.D., Topeka. Born in 1939. Graduated from the University of Kansas School of Medicine in 1965. Practices Diagnostic Roentgenology. Has served as Councilor.

G. Rex Stone, M.D., Manhattan. Born in 1929. Graduated from the University of Kansas School of Medicine in 1954. Practices General Surgery. Is now serving as Speaker.

Wayne O. Wallace, Jr., M.D., Atchison. Born in 1936. Graduated from the University of Missouri School of Medicine, Columbia, in 1965. Is in Family Practice. Is currently serving as Councilor and as Chairman of the Drug Utilization Review Committee.

Speaker

G. Rex Stone, M.D., Manhattan. Born in 1929. Graduated from the University of Kansas School of Medicine in 1954. Practices General Surgery. Is now serving as Speaker.

Vice Speaker

Franklin G. Bichlmeier, M.D., Kansas City. Born in 1933. Graduated from the University of Kansas School of Medicine in 1958. Practices Cardiovascular Surgery. Is currently serving as Chairman of the Judicial Committee and as Vice Speaker.

AMA Delegate

Lew W. Purinton, M.D., Wichita. Born in 1923. Graduated from the University of Kansas School of Medicine in 1948. Practices Internal Medicine. Is currently serving as AMA Alternate Delegate.

Kermit G. Wedel, M.D., Minneapolis. Born in 1932. Graduated from the University of Kansas School of Medicine in 1960. Is in Family Practice. Has served as Councilor. Is currently serving as AMA Alternate Delegate and as KMS President.

AMA Alternate Delegate

Ray E. Allen, M.D., Liberal. Born in 1937. Graduated from the University of Kansas School of Medicine in 1964. Practices Internal Medicine. Has served on the KMS Continuing Education Committee.

Jimmie A. Gleason, M.D., Topeka. Born in 1933. Graduated from the University of Kansas School of Medicine in 1958. Practices Obstetrics and Gynecology. Is now President Elect and has chaired the Legislative Committee for many years.

Robert P. Hudson, M.D., Kansas City. Born in 1926. Graduated from the University of Kansas School of Medicine in 1952. Practices Internal Medicine Has long been active in KMS committee work.

Linda D. Warren, M.D., Hanover. Born in 1944. Graduated from the University of Kansas School of Medicine in 1970. Is in Family Practice. Is active in AMA and KMS committee work.

Join KaMPAC

KaMPAC MEMBERS 1983

The Kansas Medical Politial Action Committee was formed by the Kansas Medical Society in 1962 in order (1) to promote and strive for the improvement of government by encouraging and stimulating physicians and others to take a more active and effective part in governmental affairs; (2) to encourage physicians and others to understand and evaluate the nature and actions of government, the significance of political issues, the records of officeholders, and the pertinent qualifications of candidates for elective office; and (3) to assist physicians and others to organize themselves for effective political action to promote civic responsibility and integrity in government.

The Kansas Medical Society has a membership of 2,884 and the Kansas Medical Auxiliary has a membership of 1,274. The following physicians and spouses have elected to become members of KaMPAC and to support "organized medicine" in its efforts to elect "friends of medicine." This listing will be expanded each month to include new KaMPAC members.

Membership Categories

Certificate	$500—C
Sustaining	100—S
Regular	60—R

Current Members

B. John Ashley, Jr., MD, R
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John O. Austin, MD, R
William S. Baker, MD, R
Marc R. Baraban, MD, R
Norman W. Berkley, MD, R
Henry P. Beugelsdijk, MD, R
F. Calvin Bigler, MD, R
Victor W. Bikales, MD, R
Rodney L. Bishop, MD, R
Paul F. Bogner, MD, R
A. Dean Burnett, MD, R
Robert L. Coleman, MD, R
Jack R. Cooper, MD, S
Thomas B. Coulter, MD, R
John E. Crary, MD, R
Peter J. Dalum, MD, R
Richard E. Davis, MD, R
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William L. Dillon, MD, R
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Robert D. Durst, Jr., MD, S
George Dyck, MD, R
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D. Mikel Elder, MD, S
Robert L. Ferguson, MD, R
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John W. Long, MD, R
Harold C. Mauck, MD, R
J. Mark Melhorn, MD
Katherine J. Melhorn, MD
Bruce Melin, MD, R
R. Michael Mendlick, MD, R
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Alex C. Mitchell, MD, R
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C. Erik Nye, MD, R
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David S. Zamierowski, MD, R

New Members

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Zeferino Arroyo, MD, R
Issara I. Ayuthia, MD, R
Robert E. Banks, MD, R
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Richard C. Carleton, MD, R
Anupong Chotimongkol, MD, R



Registration Notes

Those who have pre-registered may pick up their tickets and badges at the KMS Registration Desk. The Registration Desk will open at 8:00 a.m., Thursday, May 5.

The Holidome will hold our block of sleeping rooms only until two weeks prior to the meeting. Please contact them no later than **April 21.**

Cancellations will be refunded in full only if received by April 28. No refunds after **April 28.**

Roy B. Coffey, MD, R
John M. Cokeley, MD, R
Sharon A. Collins, MD, R
Clair C. Conard, MD, S
Thomas A. Craig, MD, R
Elsa C. Cura, MD, R
Donald D. Decker, MD, R
Herbert D. Doubek, MD, R
H. Richard Draemel, MD, R
Henry S. Dreher, MD, R
Bismarck C. D'Souza, MD, R
Glen E. Eaton, MD, R
Paul R. Fieldstone, MD, R
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Wayne L. Fowler, MD, R
F. Giles Freeman, MD, R
Fred A. Freeman, MD, R
James D. Gardner, MD, R
John H. Gilbert II, MD, R
Jimmie A. Gleason, MD, R
Donald D. Goering, MD, R
C. K. Gray, MD, R
James F. Greenwood, MD, R
Frank H. Griffith, MD, R
I. Ross Grimes, MD, R
James T. Grimes, MD, R
Marvin R. Gunn, MD, R
William D. Haire, MD, R
Howard C. Halvorson, MD, R
Stanley E. Handshy, MD
Gary Lynn Harbin, MD, R
Gene O. Harpster, MD, R
James P. Hostetter, MD, R
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Richard W. Kalbac, MD, R
Richard R. Kimball, MD, R
Steven M. Knecht, MD, R
Alan L. Kruckemyer, MD, R
George R. Learned, MD, R
George E. Liesmann, MD, R
Varden J. Loganbill, MD, R
Stanley W. Lowe, MD, R
Jack E. Lungstrum, MD, R
Ted L. Macy, MD, R
Daniel C. Martin, MD, R
Robert C. Mathews, MD, R
Jerry L. Mathis, MD, R
Walter Mau, MD, R
William H. McEachen, MD, R
Elden V. Miller, MD, R
Robert E. Miller, MD, R
Jack M. Mohler, MD, R
Merle D. Morris, MD, R
Gerald L. Mowry, MD, R
Vicente D. Narciso, MD, R
Richard H. O'Donnell, MD, S
Robert D. Olney, MD, R
Jack T. Peterson, MD, R
James E. Peterson, MD, R
Edwin D. Rathbun, MD, R
William J. Reals, MD, S
Satti S. Reddy, MD, R
Richard J. Reece, MD, R
John H. Rempel, MD, S
Mrs. John H. Rempel, S
J. M. Richardson, MD, R
David B. Robinson, MD, R
Donald C. Rorabaugh, MD, R
Larry Rotert, MD, R
Terry B. Rothstein, MD, R
J. Alan Sanders, MD, R
Steven G. Sebree, MD, R
Richard A. Siemens, MD, R
Milo G. Sloo, MD, R
David E. Smith, MD, R
Harold R. Smith, MD, R
Philip L. Stevens, MD, R
Morgan U. Stockwell, MD, R
G. Rex Stone, MD, R
Grant C. Stone, MD, R
Lawrence E. Stoskopf, MD, R
Thomas F. Taylor, MD, R
William H. Tiemann, MD, R
Roger C. Trotter, MD, R
Abdurrahman Unal, MD, R
Larry D. Vande Garde, MD, R
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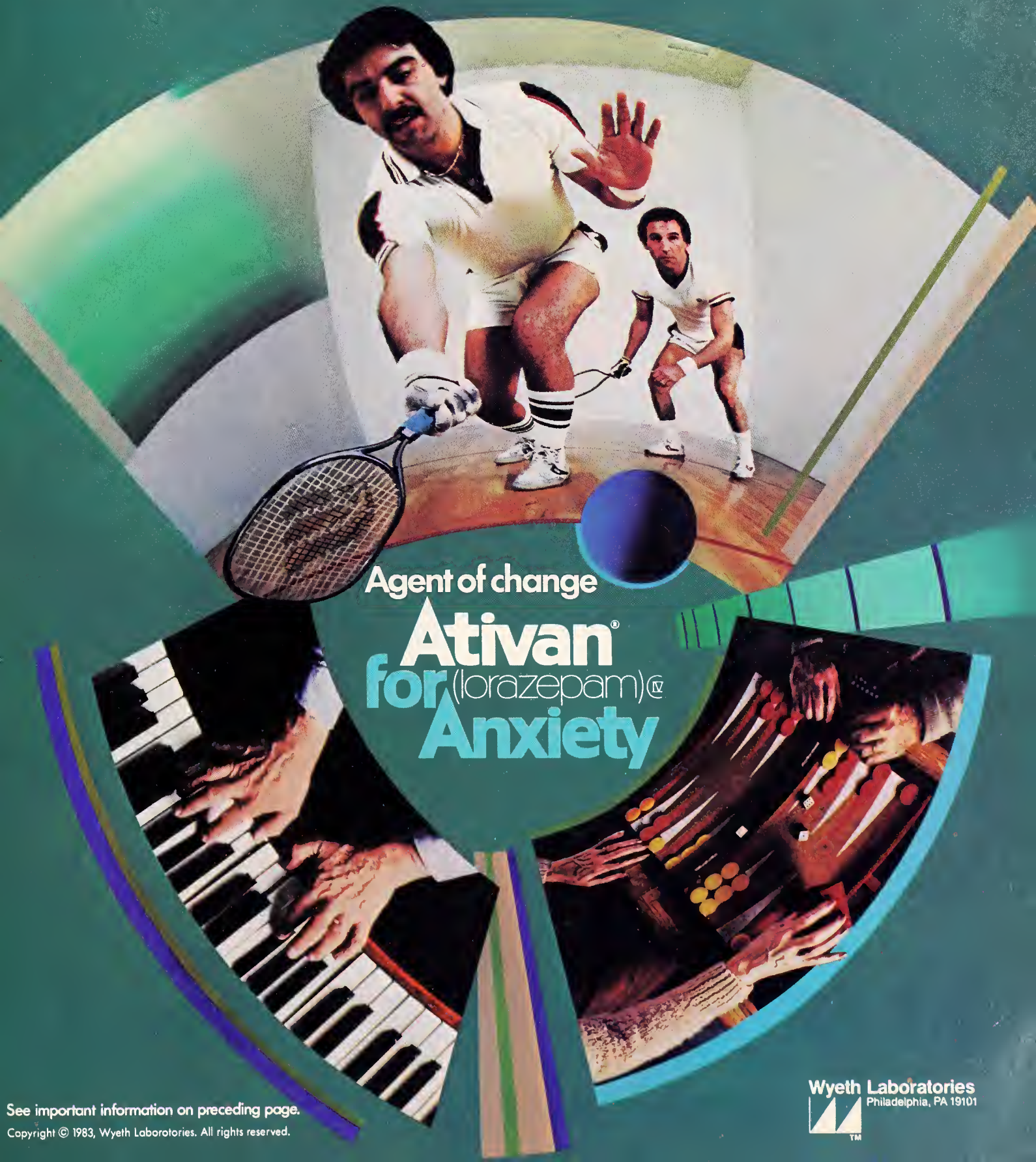
TELEPHONE NUMBERS

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Agent of change

Ativan®

for (lorazepam) IV

Anxiety

See important information on preceding page.

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NEW TABLET DESIGN 0.5 mg and 1 mg

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Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

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DOSAGE: Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.

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Welcome to Lawrence

As President of the Douglas County Medical Society, I wish to welcome the members of the Kansas Medical Society and their spouses to the annual meeting of the Kansas Medical Society and its Auxiliary, May 5-8, 1983. The meeting will be held at the Holiday Inn Holidome, Lawrence.

We have arranged a most unique and excellent program for this meeting. It is our pleasure to welcome you and act as hosts of this first meeting in Lawrence. We hope that you can join us in May.

G. B. Pees, M.D., President
Douglas County Medical Society

Hosts for the Meeting

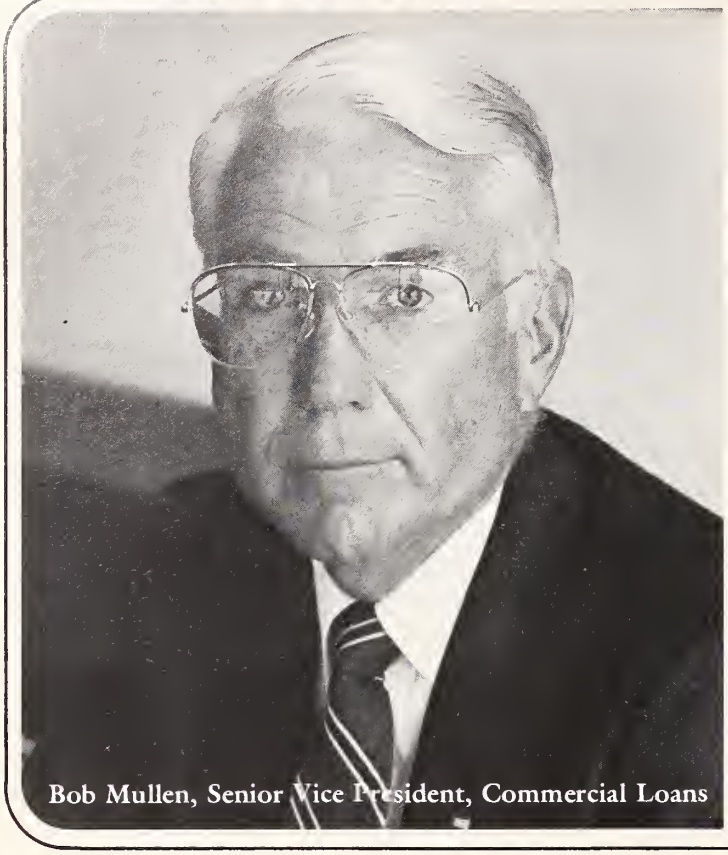


Douglas County KMS and KMSA Members Arranging the 1983 Session

General Program Committee
Gerald B. Pees, M.D., Chairman
Carol A. Moddrell, M.D. *and*
Donald W. Hatton, M.D., Scientific Program
Kenneth L. Wertzberger, M.D., Sports Day

Auxiliary Committee
Markie Bieri (Peter), Chairman
Phoebe Godwin (Phillip), Auction
Mary Lou Roberts (Richard), Courtesy
Marilyn Bittenbender (Lee) *and*
Lori Myrick (Stephen), Decorations
Kathy Bruner (Steven), Hospitality
Shirley Wilcox (Howard), Publicity
Connie Friesen (Dale), Registration
Vivian Monckton (Larry), Reservations & Finance
Barbara Learned (George), Sports Day
Shirley Reese (John), Special Arrangements

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House of Delegates

Holidome

FRIDAY — MAY 6

7:30 Registration of Delegates
9:00 First Session:
Nominations and Primary Election

SUNDAY — MAY 8

7:00 Registration of Delegates
8:00 Second Session:
Election of Officers
11:00 Council Meeting

Reference Committee

Friday, May 6, following First Session; continued at 2:00 if necessary.

John W. Young, M.D., *Shawnee Mission,* Chairman
Tell B. Copening, M.D., *Iola*
Edwin D. Rathbun, M.D., *Liberal*
David E. Street, M.D., *Wichita*
Wayne O. Wallace, Jr., M.D., *Atchison*



Annual Meeting Grants

The Kansas Medical Society is grateful for the grants and contributions received from:

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Sports Day Leaders

For the Non-Athlete and Athlete



CHRISTINE ANN AGUIAR, Ph.D.

Dr. Aguiar earned B.S. and M.S. degrees at Oklahoma universities and recently completed her doctorate at the University of Kansas. She has been certified as an Exercise Test Technologist by the American College of Sports Medicine. She is a member of several professional organizations, and has written for health related journals. She has received various student honors and is active in community service work.

Dr. Aguiar will be a leader/speaker for "Sports Day for the Non-Athlete and Athlete."

DONNA L. OSNESS, R.N., Ed.D.

Dr. Osness received her nursing education at Madison (Wisconsin) General Hospital and subsequently earned B.S., M.S., and Ed.D. degrees at the University of Kansas. At present she is Director of Health and Safety Education and Health Services for Shawnee Mission School District #512.

She has professional experience in nursing, teaching, and administration. She is active in a number of professional organizations and has participated in various civic health related activities. She has been a consultant on curriculum design for public schools, and is the author of a number of health and fitness related articles. She has been involved in research, and has been the recipient of several grants.

Dr. Osness will be a leader/speaker for "Sports Day for the Non-Athlete and Athlete."

WAYNE H. OSNESS, Ph.D.

Dr. Osness earned B.S., M.S., and Ph.D. degrees from Wisconsin universities. He currently is Professor of Physical Education; Professor of Physiology and Cell Biology; and Chairman, Department of Health, Physical Education, and Recreation at the University of Kansas. He has extensive experience in teaching, administration, and research. He is an active participant in many professional organizations and activities, and has received numerous honors and awards. He has authored a large body of published material and has made presentations to many groups worldwide. He has served as fitness consultant to several organizations.

Dr. Osness will be a leader/speaker for "Sports Day for the Non-Athlete and Athlete."

TOM R. THOMAS, Ph.D.

Dr. Thomas earned B.S., M.S., and Ph.D. degrees from various Missouri universities. He has had extensive teaching and coaching experience, and is now Associate Professor of Physical Education and Physiology and Cell Biology at the University of Kansas. He has written extensively and has made numerous presentations relating to health, fitness, and sports. He is active in several professional organizations, and has been the recipient of various honors and research grants.

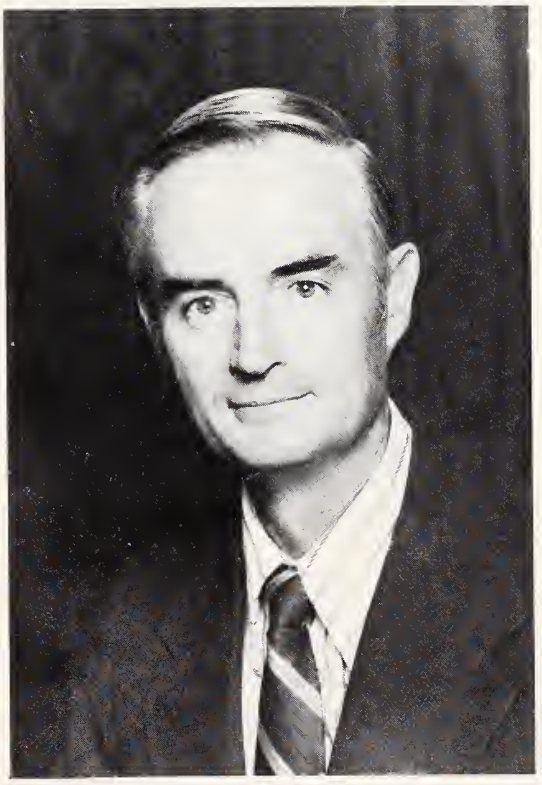
Dr. Thomas will be a leader/speaker for "Sports Day for the Non-Athlete and Athlete."

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Lawrence Area Code — 913

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Robinson Gymnasium	864-3371
Alvamar Country Club	842-1907
Lawrence Country Club	843-2938
Cedar Hill Trap Club	843-8213
Elks Club	843-3995

Distinguished Speakers



JAMES J. BERGIN, M.D.

Dr. Bergin earned his bachelor and medical degrees from Tufts University and subsequently completed several postgraduate programs. He served in the Armed Forces Medical Services. He currently is Director of Medical Education for Bethany Medical Center as well as Professor of Medicine at the University of Kansas School of Medicine-Kansas City. He is certified in Internal Medicine.

He is active in numerous professional organizations and has received various honors and awards, including the Outstanding Teacher Award from the UKSM Department of Medicine. He has written extensively for medical publications and is co-author of a book.

Dr. Bergin will discuss "Unusual Manifestations of Cancer" at the scientific session.



JOSEPH F. BOYLE, M.D.

Dr. Boyle earned his medical degree from Temple University School of Medicine and completed graduate work in New Jersey and California. He currently is in private practice in Internal Medicine in Los Angeles. He is a Diplomate of the American Board of Internal Medicine.

He was elected to the American Medical Association Board of Trustees in 1975 and has been Chairman since June 1981. He is on the faculty of the University of Southern California School of Medicine and is active in community and professional affairs. He is author of numerous papers on pulmonary and respiratory diseases.

Dr. Boyle will address the First House of Delegates.



GEORGE J. FARHA, M.D.

Dr. Farha earned his medical degree from Tulane University and subsequently completed graduate training in Wichita and Boston. He is certified by the American Board of Surgery and currently practices General Surgery in Wichita. He also is a member of the medical faculties of the University of Kansas School of Medicine-Wichita and St. Francis Regional Medical Center.

He has had extensive administrative experience and is active in a number of professional and civic organizations. He has written numerous articles for medical publications.

Dr. Farha will discuss the "Surgical Approach to Gastrointestinal Malignancy" at the scientific session, and will also participate in the panel discussion.



JIMMIE A. GLEASON, M.D.

Dr. Gleason earned his M.D. degree from the University of Kansas School of Medicine. He currently practices Obstetrics and Gynecology in Topeka.

He has been active in professional organizations, and is now KMS President Elect.

Dr. Gleason will address the scientific session on "Diagnostic Gynecology — What to do with the Abnormal Pap Smear."

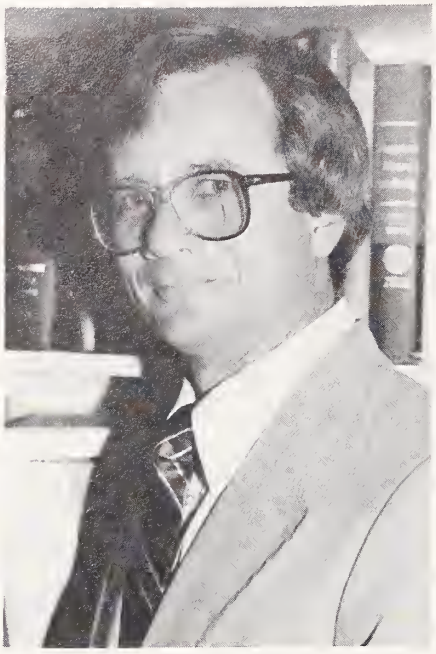


JAVIER F. MAGRINA, M.D.

Educated primarily in Spain, Dr. Magrina completed graduate training at Mayo's and at the University of Kansas School of Medicine-Kansas City. He currently is Acting Director, Division of Gynecologic Oncology, Department of Obstetrics and Gynecology at both UKSM-KC and at the University of Missouri in Kansas City. He is certified in Obstetrics and Gynecology and in Gynecologic Oncology.

He is active in several professional organizations and has received a number of honors and awards. He has written extensively for medical publications and has translated medical material from English to Spanish. He also has made numerous professional presentations.

Dr. Magrina's address for the scientific session will be on "Treatment of GYN Malignancies."

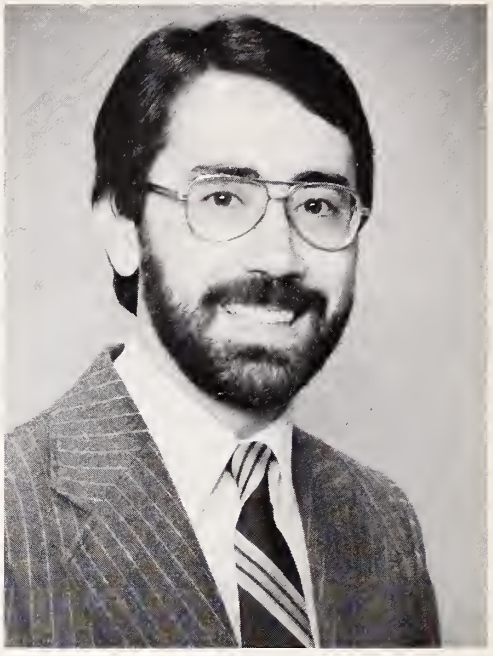


RICHARD MEIDINGER, M.D.

Dr. Meidinger earned B.A. and M.D. degrees from the University of Kansas. Following internship, he served in the United States Air Force for two years. Currently he is in private practice of Radiology and Nuclear Medicine in Topeka. He also is Clinical Associate in Nuclear Medicine and Diagnostic Radiology at UKSM-KC and serves as diagnostic radiology consultant for a number of area hospitals. He is certified in Diagnostic Radiology, Nuclear Medicine, and Nuclear Radiology.

He is active in professional organizations and has written for medical publications. He has extensive research experience.

Dr. Meidinger will address the scientific session on "Diagnostic Radiology — The Place of CT Scanning and Sonography" and will participate in the panel discussion.

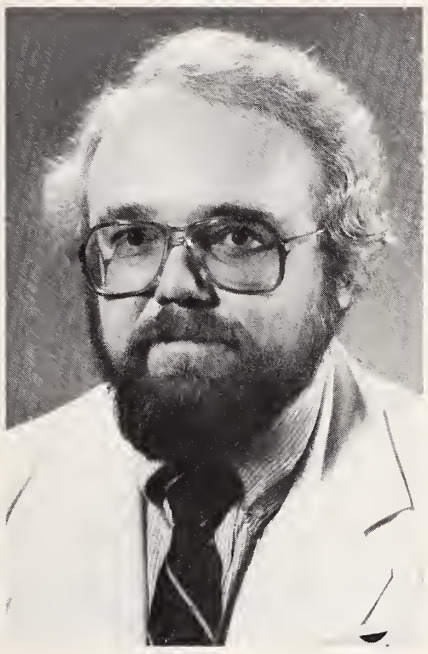


JORGE C. PARADELO, M.D.

Dr. Paradelo was educated in Argentina and has completed extensive graduate training in Argentina and the United States. He is currently in the private practice of Radiation Oncology in Kansas City, Missouri. He is certified in Therapeutic Radiology.

He is active in professional organizations and has written for medical journals and books.

Dr. Paradelo will discuss "Advances in Radiation Oncology" at the scientific session.



RONALD L. STEPHENS, M.D.

Dr. Stephens earned his medical degree from the University of Kansas School of Medicine, where he currently is Professor of Medicine and Director, Division of Clinical Oncology. He is certified in Internal Medicine and Medical Oncology.

He is active in a variety of professional organizations and activities, and has written extensively for medical publications.

Dr. Stephens' presentation to the scientific session will be entitled "Medical Emergencies in the Cancer Patient." He will also participate in the panel discussion.



LAWRENCE L. TRETBAR, M.D.

Dr. Tretbar earned his medical degree from the University of Kansas School of Medicine in 1960 and completed graduate programs in Cleveland and London. He is now in private practice of General Surgery and is a member of the surgical faculties of the Universities of Kansas and Missouri Schools of Medicine in Kansas City. He is certified by the American Boards of Surgery and Medical Examiners.

He has been director of medical and surgical education at Bethany Medical Center, Kansas City. He was primary medical advisor for the Medical Skills Library project of the American College of Physicians. He has published and presented a number of papers, and has assisted in the production of several medical teaching films.

Dr. Tretbar will speak to the scientific session on "Surgical Approaches to the Complications of Malignancy," and will also participate in the panel discussion.



TRIBHAWAN S. VATS, M.D.

Dr. Vats was educated in India and subsequently completed several postgraduate programs in Connecticut, Arizona, and New York. He currently is Professor of Pediatrics and Section Chief/Pediatric Hematology-Oncology at the University of Kansas School of Medicine-Kansas City. He also serves as Director of Student Education in Pediatrics. He is certified in Pediatrics and in Pediatric Hematology/Oncology.

He is active in a number of professional organizations and has received various honors, awards, and research grants. He has written extensively for medical publications and books.

Dr. Vats will address the scientific session on "Non-Hematologic Malignancies of Childhood."



STANLEY J. VOGEL, M.D.

Dr. Vogel earned his medical degree, with Honors in Internal Medicine, from Washington University School of Medicine, and completed graduate programs in Missouri and Michigan. He currently is in the private practice of Internal Medicine in Topeka and is an Instructor in the Department of Internal Medicine at the University of Kansas School of Medicine-Kansas City. He served in the United States Army Medical Corps for two years. He is certified as a Medical Examiner, in Internal Medicine, and in Medical Oncology.

He participates in professional, civic, and research activities and has written for medical publications.

Dr. Vogel will discuss "Cancer Screening — Fact or Fiction" at the scientific session, and will participate in the panel discussion.



HOWARD N. WARD, M.D., J.D.

Dr. Ward received his medical education at the University of Illinois and Northwestern University Medical School; he also earned a law degree from Washburn University School of Law. He earned scholastic honors at all three institutions. He currently practices hematology in Topeka and is Adjunct Assistant Professor of Law at Washburn University School of Law.

He has served in the United States Air Force Medical Corps. He is certified in Internal Medicine, Hematology and Medical Oncology, and is active in numerous medical, legal, and civic organizations. He has written for various medical publications and has made presentations to professional groups.

Dr. Ward will address the general luncheon during the scientific session; his topic will be "Euthanasia."

TELEPHONE NUMBERS

Lawrence Area Code — 913

Holidome	841-7077
Robinson Gymnasium	864-3371
Alvamar Country Club	842-1907
Lawrence Country Club	843-2938
Cedar Hill Trap Club	843-8213
Elks Club	843-3995



Doctor and Mrs. Kermit G. Wedel extend a personal invitation to members of the Society and their guests to attend the installation festivities on Saturday, May 7, 1983. Installation of the new KMS President will be at 5:30 PM in the Regency Suite followed at 6:30 PM by the President's Reception with champagne and hors d'oeuvres.



KANSAS MEDICAL SOCIETY

PROGRAM INFORMATION

Thursday, May 5

Traditional Sports Activities
Alvamar Country Club

11:00 a.m. GOLF — Fees: $15.00 per person
$14.00 per golf cart

2:00 p.m. TENNIS — Alvamar Racquet and Tennis Club
Fee: $10.00 per person
Doubles and Mixed Doubles — Class A, B,C,

2:00 p.m. TRAP SHOOT — Cedar Hill Trap Club
(Offered only if adequate number of registrants received by April 21)

Sports Day
For the Non-Athlete and Athlete

8:45-9:00 a.m. Buses leave Holidome — tour across campus to Robinson Gymnasium. (First 33 to board bus, ride on "Big Blue KU" bus; humorous tour dialogue by the next Auxiliary President, Diane Sanders (Alan). Dress in stretchable exercise clothes (preferably no pant zippers, button or belt buckles) and rubber soled, lace up shoes. Carry clean leisure clothes for change after activities. Security for valuables will be provided.

9:00-9:15 a.m. Unload at Robinson. Auxiliary tour guides will direct participants to security locker area, then to general meeting gymnasium (informal seating on gym floor). Registration required to receive Category I Credit.

9:15-9:30 a.m. "FITNESS WELCOME TO KMS/KMSA" by KU Chancellor ***Gene Budig*** and ***Wayne H. Osness, Ph.D.,*** Chairman of Department of Health, Physical Education and Recreation, University of Kansas.

9:30-10:00 a.m. Lecture: "Necessity of Correct Procedures for Stretching." Lecture and participation.
Christine A. Aguiar, Ph.D., Exercise Physiology.

10:00-10:45 a.m. Activities. (Activity fun, also educational approach by graduate student leaders). Choice of one activity. Please check on Sports Day registration form:
Swim, aerobic/recreational
Physical development (weight room)
Racquetball
Tennis (rackets can be provided)
Aerobic dance, beginner and experienced
Square Dance
Lifetime sports room, table tennis or badminton
Jog, with Dr. Wayne Osness, on scenic tour around campus or in Allen Fieldhouse if rain
Walk, for those with physical problems — to be adjusted to situations and people — possibly around lower flat campus or on Hershberger rubber track in Allen Fieldhouse.
Individualized physical fitness profiles ($15.00 for individual to be paid at time of registration for Sports Day or during activity. First five participants to send money and pre-registration have choice of evaluation time, 10:00-10:45 or 1:15. All others after 1:15. Limited to 30 participants).

10:45-11:15 a.m. Shower and dress. Free clean towels and hair drying facilities.

11:15-12:00 noon Food demonstration, "A Food for Fitness" and "informal" lunch. Cooked and served by Douglas County Medical Auxiliary and Society. Free recipe booklets.

12:00-1:00 p.m. Lectures:
Tom R. Thomas, Ph.D. Basic exercise physiology in relation to the laboratory. Review of current literature relating to biochemical implications of exercise.

Wayne H. Osness, Ph.D. Evaluation of physiological performance and development of exercise prescription for individuals contemplating exercise intervention. Current research relating to this topic.

Donna L. Osness, R.N., Ed.D. Current curricular activities relating to health enhancement and life style intervention. Sunflower Project in Kansas schools and related programs.

1:00 p.m. Program Evaluation (for CME certification)

1:15 p.m. Buses return to Holidome.

1:15 p.m. Extra time for physical fitness profiles — as needed. (Late transportation back to Holidome by Holidome bus or Douglas County Auxiliary members).

Fee: $5.00 per person (includes lectures, activities and luncheon).
Kansas Governor's Council on Fitness T-shirt — $4.00
Individualized physical fitness Profile — $15.00

"As an organization accredited for continuing medical education, the University of Kansas College of Health Sciences, Division of Health Care Outreach and Continuing Education designates this activity as meeting the criteria for 3 credit hours in Category I of the Physician's Recognition Award of the American Medical Association."

6:00 p.m. Social Hour (Cash Bar) Elks Club
7:00 p.m. Sports Banquet — Fee: $15.00 per person
Speaker: ***Wayne H. Osness, Ph.D.***
Topic: "Personal Fitness Management"

TELEPHONE NUMBERS

Lawrence Area Code — 913

Holidome	841-7077
Robinson Gymnasium	864-3371
Alvamar Country Club	842-1907
Lawrence Country Club	843-2938
Cedar Hill Trap Club	843-8213
Elks Club	843-3995



Friday, May 6

Holiday Inn — Holidome

7:00 a.m. Kansas Foundation for Medical Care Board of Directors — Brazilian D

7:30 a.m. Registration Opens — Lobby

9:00 a.m. First House of Delegates and Reference Committee — Brazilian A & B

12:30 p.m. Delegates Luncheon — Brazilian C & D
Fee: $7.25 per person

2:00 p.m. Reference Committee (Continued if necessary — Brazilian A & B

2:00 p.m. AMA Tele-Net — The Future for Medicine

4:00 p.m. KaMPAC Hospitality Suite — Brazilian C
Cocktails & Hors d'Oeuvres (complimentary)

4:00 p.m. Kansas Society of Internal Medicine Reception — Regency D11

5:30 p.m. Social Hour (Cash Bar) — Regency A, B, C, & D

6:30 p.m. Dinner Sponsored by Kansas Medical Society Auxiliary. Followed by AMA-ERF Auction and Prairie Party. Fee: $16.50 per person

Saturday, May 7

7:30 a.m. Registration Opens — Lobby
7:30 a.m. Past Presidents Breakfast — Regency D 1
8:00 a.m. Scientific Program — "Cancer in Kansas — 1983" — Brazilian A & B
Objectives:
1. The physician participants will be able to design a reasonable screening program for identifying cancer in their patients.
2. The physician participants will be able to identify the best radiologic procedures to evaluate various organ cancers.
3. The physician participants will be able to draw a logical algorithm for evaluation of the abnormal Pap.
4. The physician participants will be able to identify medical emergencies commonly associated with cancer.
5. Resource persons in the state of Kansas, with experience in dealing with various types of cancer, will be identified.
6. Physician participants will be able to identify unusual conditions associated with cancer.

8:00-8:15 a.m. Welcome and Announcements
8:15-8:45 a.m. "Cancer Screening — Fact or Fiction" ***Stanley J. Vogel, M.D.,*** Topeka
8:45-9:15 a.m. "Diagnostic Radiology — the Place of Ct Scanning and Sonography"
Richard Meidinger, M.D., Topeka
9:15-9:45 a.m. "Diagnostic Gynecology — What to do With the Abnormal Pap Smear"
Jimmie A. Gleason, M.D., Topeka
9:45-10:15 a.m. "Surgical Approach to Gastrointestinal Malignancy"
George J. Farha, M.D., Wichita
10:15-10:30 a.m. Coffee Break
10:30-11:00 a.m. "Medical Emergencies in the Cancer Patient"
Ronald L. Stephens, M.D., Kansas City
11:00-11:30 a.m. "Surgical Approach to the Complications of Malignancy"
Lawrence L. Tretbar, M.D., Shawnee Mission
11:30-12:00 noon Panel Discussion
Drs. Vogel, Meidinger, Gleason, Farha, Stephens & Tretbar
12:00-1:30 p.m. General Luncheon — Brazilian C & D
Speaker: ***Howard N. Ward, M.D.,*** Topeka
Topic: "Euthanasia"
1:30-2:00 p.m. "Non-Hematologic Malignancies of Childhood"
T. S. Vats, M.D., Kansas City
2:00-2:30 p.m. "Advances in Radiation Oncology"
Jorge C. Paradelo, M.D., Kansas City, MO
2:30-3:00 p.m. "Treatment of GYN Malignancies"
Javier F. Magrina, M.D., Kansas City
3:00-3:15 p.m. Coffee Break
3:15-3:45 p.m. "Pediatric Leukemia"
T. S. Vats, M.D., Kansas City
3:45-4:15 p.m. "Unusual Manifestations of Cancer"
James J. Bergin, M.D., Kansas City

Fee: $25.00 per person (includes lunch)

"As an organization accredited for continuing medical education, the Lawrence Memorial Hospital designates this activity as meeting the criteria for 6 credit hours in Category I of the Physician's Recognition Award of the American Medical Association."
This program has been reviewed and is acceptable for 6 prescribed hours by the American Academy of Family Physicians.

11:30 a.m. Kansas OB-Gyn Society — Regency A
Luncheon and Business Meeting
11:30 a.m. Kansas Society of Pathology — Regency D11
Luncheon and Business Meeting
11:30 a.m. Kansas Society of Anesthesiology — Regency B
Luncheon and Business Meeting
11:30 a.m. Section on Ear, Nose & throat — Regency D1
Luncheon and Business Meeting
12:00 noon Therapeutic Radiologists Society — Ramada Inn
5:30 p.m. Installation of Kansas Medical Society President — Regency A, B, C & D
6:30-8:30 p.m. Kansas Medical Society President's Reception
Champagne and Hors d'Oeuvres



Sunday, May 8

7:00 a.m. Registration Opens — Lobby
7:00 a.m. Delegates Breakfast — Regency A & B
Fee: $5.25 per person
8:00 a.m. Second House of Delegates — Brazilian A & B
11:00 a.m. Council — Regency A
11:00 a.m. Kansas Urological Society — Regency D1

Specialty Societies

Friday, May 6
4:00 Kansas Society of Internal Medicine
Regency D11
Reception

Saturday, May 7
11:30 Kansas OB-GYN Society
Regency A
Luncheon and Business Meeting
11:30 Kansas Society of Pathology
Regency D11
Luncheon and Business Meeting
11:30 Kansas Society of Anesthesiology
Regency B
Luncheon and Business Meeting
11:30 Section on Ear, Nose & Throat
Regency D1
Luncheon and Business Meeting
12:00 Therapeutic Radiologists
Ramada Inn
Luncheon and Business Meeting
12:00 Section on Nuclear Medicine
Ramada Inn
Luncheon and Business Meeting

MEDICAL ASSISTANTS

Many physicians are unaware of the fact that for more than 25 years there has been a movement to assure a continuing source of personnel specifically trained to work in the medical office setting or other health facility. This movement, spearheaded by the American Association of Medical Assistants (AAMA), has accomplished these goals:

- Founded a tri-level educational organization which now has 47 state affiliates and 600 local chapters.
- Established the AAMA Endowment as a public foundation, and the Maxine Williams Scholarship Fund to help young people to attain medical assistant training.
- Created the Certifying Board and pioneered the certification program which was the first in the country to establish and recognize professional standards for medical assistants.
- Developed curriculum standards for the training of medical assistants, and established AAMA's Curriculum Review Board, which collaborates with the American Medical Association in accreditation of postsecondary educational programs by the Committee on Allied Health Education and Accreditation. AAMA is recognized by both the U.S. Office of Education and the Council on Postsecondary Accreditation as an official accreditating agency.
- Created an Education Council which functions annually to coordinate the educational activities of the organization. In 1976, the Continuing Education Committee officially launched the Continuing Education Unit Program.
- Developed two guided study courses: Anatomy, Terminology and Physiology; and the Humanistic Medical Assistant.
- In April 1980, AAMA received formal recognition of the medical assisting occupation from the U.S. Department of Health, Education, and Welfare.

There are approximately 143 AAMA-approved training programs in community colleges and commercial schools. These programs produce about 5,000 new medical assistants annually. It is a good career for a mature person, but it is work that appeals to people of all ages, since it has many job satisfactions associated with it. AAMA local chapters, state societies, and the national organization regularly present educational programs designed to expand members' knowledge and skills for those who are interested in continuing their education.

AAMA, with its ever-increasing stature, its unlimited opportunities for self-improvement, its high ideals and objectives of education and increased efficiency, is indeed an organization worth talking about. We must make others see that membership in AAMA is something to be highly prized.

Now the time has come to ask, "Where are we?" and "How are things going?" Our major problem continues to be economic: How do we maintain funding for such programs as continuing education and certification at the level of quality AAMA members and our physician-employers have come to expect? In a period of continuously rising costs, it is apparent that we have arrived at a plateau of income from other sources, and we are hard pressed to consolidate the gains we have made. Thus the struggle goes on.

A 1977 survey revealed that 46 per cent of physician-employers paid dues on behalf of their employees. With the AAMA tri-level membership, dues are a substantial outlay for some medical assistants. At least 1,200 physicians presently serving as advisors to medical assistants groups at all levels would agree that paying dues is a good investment for a physician, and it is tax deductible. Joining AAMA enables the medical assistant to exchange ideas and share information as well as participate in the educational programs thus increasing the assistant's ability to serve the physician-employer and the public.

The value of an association like AAMA is immeasurable. There is nothing like collective wisdom, talent, and dedication of individuals working together for the purpose of the advancement of the profession and its members. It is hoped that physicians will recognize medical assistants as qualified professionals who are dedicated not only to helping make medical practices function efficiently but also to the patients' well-being.

As medical technology continues to grow, efficient and conscientious medical assistants will play an expanding role in your practice. Help them to stay abreast of developments and to hone their skills by encouraging them to join AAMA and by paying their dues. With your support, AAMA can help them to feel the same commitment to their profession as you do to yours. Since the majority of AAMA programs stress practical application, you will see an immediate impact on your practice.

Frances M. Nastav, *President*
AAMA-Kansas Society

KaMPAC

The purposes of KaMPAC are:

- To promote and strive for the improvement of government by encouraging and stimulating physicians and others to take a more active and effective part in governmental affairs;
- To encourage physicians and others to understand and evaluate the nature and actions of government, the significance of political issues, the records of officeholders, and the pertinent qualifications of candidates for elective office; and
- To assist physicians and others to organize themselves for effective political action to promote civic responsibility and integrity in government.

The KaMPAC Board of Directors is composed of the following members:

Roger D. Warren, M.D., Chairman, *Hanover*
John P. Brockhouse, M.D., *Emporia*
Jack R. Cooper, M.D., *Shawnee Mission*
Vernon W. Filley, M.D., *Pratt*
Twila Flowers (Clell), *Wichita*
Paul D. Johnson, M.D., *Leavenworth*
R. Rex Lee, M.D., *Wichita*
Dot Meyer (Warren), *Wichita*
Betty Moore (Robert), *Caney*
James J. Muehlberger, M.D., *Shawnee Mission*
Richard H. O'Donnell, M.D., *Clay Center*
Daniel N. Pauls, M.D., *Parsons*
Forrest A. Pommerenke, M.D., *DeSoto*
Katherine Pyle (Lucien), *Topeka*
Charles S. Reeves, M.D., *Fort Scott*
Ann Rempel (John), *Wichita*
Alex Scott, M.D., *Junction City*
Thomas F. Taylor, M.D., *Salina*

KaMPAC membership in 1982 totaled 496, the highest since 1977. In the years 1981 and 1982, KaMPAC raised a total of $49,515, or an average of $17.23 per physician in Kansas. Of the total amount raised, $29,500 went for candidate support, $17,430 transferred memberships to AMPAC, leaving a balance of $2,585.

In the 1982 elections, KaMPAC contributed a total of $29,500 to 109 primary and general election campaigns. Ninety-six of the 109 candidates won their elections for an overall winning average of 88 per cent.

Roger Warren, M.D., Chairman, visited many of the Council Districts and discussed the importance of involvement in political action. Local contact persons were established in each county medical society for additional input from the local level.

The Board, in conjunction with AMPAC, is planning to sponsor a 1½ day educational seminar on political action at the 1984 Annual Meeting in Hutchinson.

KANSAS FOUNDATION FOR MEDICAL CARE, INC.

Medical peer review in Kansas truly came of age during the last year. The Kansas Foundation for Medical Care, Inc. (KFMC), the state's only Professional Standards Review Organization (PSRO), celebrated its most successful operation in the decade since its inception.

The Health Care Financing Administration credited the Foundation with saving more than 80,000 documented hospital days while promoting quality practice. KFMC has acquired a national reputation for assuring quality health care at a reasonable cost.

Quality Assurance

All 144 Kansas acute care hospitals participated in two or more areawide quality assurance studies.

- The antibiotic prophylaxis study demonstrated a need for improved timing of administration.
- The nosocomial urinary tract infection (UTI) study showed that one-third of UTIs identified were nosocomial. It demonstrated a need for better infection prevention protocol.
- The cerebrovascular accident (CVA) study revealed a need for earlier intervention with CVA patients and a need for discharge planning.
- The back pain study indicated that out-patient services could be better utilized for initial treatment of patients with back pain. The amounts of narcotics used were less than anticipated.

Every Medicare patient's record is screened for three quality indicators: Readmission; use of respiratory therapy, with emphasis on intermittent positive pressure breathing; and anesthesia documentation. Hospitals with aberrant patterns in these areas are closely monitored for compliance with standards.

Utilization Review

In an effort to achieve the greatest impact for a given cost of review, KFMC used its extensive Medicare data to focus review on problem areas. Called "data-based review," the method reserves the intensive "concurrent" technique for providers with aberrant practice patterns.

Until last year, the Foundation was funded entire-

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ly by a federal grant. The first review contract was with the Kansas Department of Social and Rehabilitation Services for Medicaid patients. Delegated concurrent review was implemented in five large counties, and non-delegated review began in two large counties. The remainder of the state was served by a novel system of non-delegated review by telephone from the Topeka office.

Private industry and insurance companies have actively sought review agreements with KFMC during the past year. A coalition of health care purchasers in Kansas City has signed agreements with KFMC and the Missouri-side PSRO for concurrent review of all private patients in that city's hospitals. The program, called Midlands Medical Review Plan, Inc., is expected to phase in this spring. When fully operational, it will cover 50,000 Kansas patients annually. The Foundation is negotiating with a score of businesses and insurance companies for private review throughout Kansas.

Two new varieties of review are now being explored. One is screening for hospital procedures that could have been performed on an out-patient basis. The other is examination of patient records and billing forms to assure that services charged were actually ordered and delivered.

Last year the Foundation broadened its scope to include review of three psychiatric facilities.

Data

KFMC now operates its own in-house computer system. Programmed for revealing practice patterns, the system stores and analyzes a vast amount of historical data.

Reports produced automatically by computer include:

- Each hospital's top 20 diagnoses, procedures and diagnostic related groups (DRGs), as well as the average length of stay.
- Hospital profile: statistics on a variety of utilization parameters.
- Quality assurance profile for readmissions, use of respiratory therapy, anesthesia documentation.
- Ancillary services profile: X-rays, EKGs, and laboratory work.

Perhaps the most useful statistic in the new reports is the "utilization index," which compares average length of stay for patients of individual providers with the statewide average, equated for case mix.

As a special service to hospitals, KFMC is providing, at cost, a reevaluation of DRGs, which can be used by hospitals to maximize their reimbursement under the proposed Medicare prospective payment system.

Because the Foundation now associates a unique physician identification number with each case, the data department can generate a utilization profile by physician, just as it has by hospital. The implications for physician education are great.

Education and Public Relations

KFMC staff conducted ten regional meetings to acquaint the provider community with changes in Medicare and Medicaid review programs.

Foundation members were encouraged to write to their elected representatives in support of peer review legislation.

New publications include an introductory brochure for private review clients and the first KFMC Annual Report. A ten-minute audio-visual presentation is quite helpful in introducing peer review.

A speakers' bureau is being formed to help acquaint hospital medical staffs and civic organizations with Foundation activities.

In March, the board, committees, and staff of KFMC held their first retreat in Kansas City. The educational exposure from that convention will inspire dedication to peer review for some time.

Looking Ahead

The Tax Equity and Fiscal Responsibility Act (TEFRA) included legislation to replace PSROs with Peer Review Organizations (PROs), with which the government will contract for Medicare review. KFMC firmly expects to win that contract in Kansas.

The Foundation hopes to serve its members during the coming year by becoming accredited to provide Category I CME credits.

This is an exciting time of growth and change for KFMC. With the continued support of Society members, the Foundation will meet the challenges that lie ahead.

GOOD NEWS!

You can **now** get our non-cancellable, guaranteed renewable disability plan at reduced premium rates and non-smoker rates.

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Committee REPORTS

COGNITIVE MEDICINE COMMITTEE

The committee is composed of the following members:

Joseph D. Sargent, M.D., Chairman, *Topeka*
Maurice R. Cashman, Jr., M.D., *Topeka*
John A. Segerson, M.D., *Topeka*
Arthur D. Snow, Jr., M.D., *Shawnee Mission*
G. Rex Stone, M.D., *Manhattan*
Thomas F. Taylor, M.D., *Salina*
Ralph H. Weber II, M.D., *Salina*

During the past year, the committee reviewed the issue of cognitive and procedural services and their effects on the costs of health care, distribution of physicians by specialty, and the perception that medical care has become impersonal.

The committee concurs with the American Society of Internal Medicine definition of cognitive services as follows:

> Physicians' cognitive services involve the application, based on relevant knowledge and experience, of such skills as data gathering and analysis, planning, management, decision-making, and judgment relating to the prevention, diagnosis and treatment of health problems, and communication of such information to the patient.

The committee reviewed existing utilization review programs and research on second surgical opinion programs and their effects on the quality of care. Utilization review programs include those of the Kansas Foundation for Medical Care, Medical Society of Sedgwick County Foundation for Medical Care, Blue Cross-Blue Shield, and the review program for Title XIX patients conducted by the Kansas Foundation for Medical Care. The committee supports effective utilization review programs that help to ensure the maintenance of quality of care.

Recently, the Health Care Financing Administration announced that Medicare contractors will be required to use AMA-Current Procedural Terminology (CPT) when they convert their claims processing systems. State Medicaid programs will be encouraged to convert to AMA-CPT also. This move was supported by the committee and by Kermit Wedel, M.D., President of KMS. The committee will submit a resolution informing KMS members of this and encouraging all third-party fiscal agents to implement AMA-CPT as soon as possible.

The committee would encourage the KMS Executive Committee to recognize and continue to address the value of cognitive skills as they address quality of care issues. In addition, physicians whose practices primarily involve the provision of cognitive services should be encouraged to serve on KMS committees. It is recommended that information about education and research on cognitive services be publicized where appropriate. The committee decided there was no need at present to continue as an organized committee and will dissolve as an *ad hoc* committee following the KMS State Meeting.

CONTINUING MEDICAL EDUCATION COMMITTEE

The purpose of the committee is to survey and accredit local institutions and organizations for Category I credit. The committee also provides information and consultation to organizations to help improve their programs. A regular listing of all Category I programs in Kansas is also published.

The committee is composed of the following members:

Erwin T. Janssen, M.D. Chairman, *Topeka*
Steven E. Barker, M.D., *Minneapolis*
James J. Bergin, M.D., *Kansas City*
F. Calvin Bigler, M.D., *Garden City*
Robert W. Brown, M.D., *Salina*
Louis M. Culp, M.D., *Kansas City*
Kailash Dave, Ph.D., *Parsons*

James D. Gardner, M.D., *Manhattan*
L. William Halling, M.D., *Hays*
Wilmer A. Harms, M.D., *Halstead*
Donald W. Hatton, M.D., *Lawrence*
Glen C. Hutchison, M.D., *Hays*
Merlin G. Kirby, M.D., *Great Bend*
Joseph C. Meek, Jr., M.D., *Kansas City*
Cesar V. Menez, M.D., *Shawnee Mission*
John Neuenschwander, M.D., *Hoxie*
G. William Nice, M.D., *Topeka*
Lew W. Purinton, M.D., *Wichita*
Ralph R. Reed, M.D., *Lawrence*
Jesse D. Rising, M.D., *Kansas City*
Maxwell Sloop, *Halstead*
Maurice M. Tinterow, M.D., *Wichita*
Ted Warren, Ph.D., *Topeka*
James W. Wiggs, M.D., *Great Bend*

This year seven hospitals have been surveyed or resurveyed. Three hospitals were granted accreditation, action was deferred on two hospitals pending receipt of further information, and the balance will be considered at the April meeting. Accreditation for one hospital was dropped at their request. The accreditation survey fee was increased from $150 to $650, and an annual fee of $200 was instituted to more adequately cover the costs involved in the program. A revised survey form was adopted.

The committee is planning an educational conference for Directors of Medical Education in October 1983. The conference will provide an opportunity for sharing information among accredited organizations and will also provide information about a new set of Essentials for Accreditation.

Efforts are being made to work with the Kansas Foundation for Medical Care and other statewide organizations to identify potential educational topics for distribution among the accredited organizations.

INSURANCE AFFAIRS COMMITTEE

The committee is composed of the following members:

Ralph H. Baehr, M.D., Chairman, *Topeka*
Richard J. Cummings, M.D., *Wichita*
Ann Gorrell (Ned), *Parsons*
Tom E. Kendall, M.D., *Wichita*
Mary King (William), *Great Bend*
William R. Lentz, M.D., *Topeka*
Lowell M. Rhodes, M.D., *Wichita*
Alex Scott, M.D., *Junction City*
Newton C. Smith, M.D., *Arkansas City*
Nancy J. Wing, M.D., *Topeka*

During the past year, the committee reviewed existing member benefit programs endorsed by the KMS and considered additional programs for endorsement.

The committee recommends continued endorsement of the following programs:

- Workers Compensation Program: Dodson Insurance Group — 1982 premium savings were 37 per cent.
- Disability Income Program — Washington National Insurance Company.
- Health Insurance — Blue Cross-Blue Shield.
- Accidental Death and Dismemberment — Woodsmall, Frick and Innis.
- Collections — I.C. System.
- Car Rental — Hertz Corporation: 15 per cent discount on basic rate.

The committee considered the issue of requests from insurance companies for appropriateness review of various cases. The recommendation was made to forward all such requests to the Kansas Foundation for Medical Care.

The committee studied a proposal for a hospital daily benefit insurance program and decided not to offer such a program to KMS members because national organizations currently offer better programs. In addition to endorsing the Hertz Corporation for car rentals, the committee recommends endorsement of the Avis Corporation. Rate discounts are the same for both companies.

Questions were raised about the issue of Blue Shield policy on payment for concurrent care. The committee met with a Blue Shield representative and discussed mechanisms for simplifying the documentation and paperwork required to justify payment in concurrent care situations. Blue Shield agreed to consider the mechanisms discussed.

The committee reviewed information on the Oklahoma State Medical Association Professional Liability and Health Insurance Program. The committee recommends that the information be filed and that the current system for professional liability insurance continue.

JUDICIAL COMMITTEE

The purposes of the committee are to work actively to assure that colleague physicians are of the highest character and professional ability, to protect patients from physicians who are deficient in moral character or professional competence, to assist component medical societies in establishing a due pro-

cess mechanism for handling complaints, and to handle complaints that can't be resolved at the local level.

The committee is composed of the following members:

Franklin G. Bichlmeier, M.D., Chairman, *Kansas City*
Caryl Bichlmeier (Franklin), *Kansas City*
Joy V. Bliss, M.D., *Olathe*
Maurice R. Cashman, Jr., M.D., *Topeka*
Tell B. Copening, M.D., *Iola*
Richard H. Egelhof, M.D., *Wichita*
Jimmie A. Gleason, M.D., *Topeka*
Herman H. Jones, Jr., M.D., *Kansas City*
Ward A. McClanahan, M.D., *Wichita*
John H. Rempel, M.D., *Wichita*

Activities this year have consisted primarily of referring complaints to local component medical societies for consideration. This year the majority of complaints concerned appropriateness of care issues. Very few concerned excessive fees.

In April the committee will consider complaints arising at the Kansas State Penitentiary including one possibly requiring a joint effort of the Impaired Physician Committee and the Judicial Committee.

KANSAS MEDICAL SOCIETY/KANSAS HOSPITAL ASSOCIATION LIAISON COMMITTEE

The purpose of the committee is to provide an ongoing liaison between Kansas Medical Society (KMS) and Kansas Hospital Association (KHA). The committee addresses issues of common interest and concern to both organizations.

The committee is composed of the following members:

D. Cramer Reed, M.D., Chairman, *Wichita*
David W. Bouda, M.D., *Hutchinson*
James G. Bridgens, M.D., *Shawnee Mission*
K. William Bruner, Jr., M.D., *Wichita*
Richard B. Darr, M.D., *Kansas City*
Donald D. Goering, M.D., *Coldwater*
Lunetta M. Pearce, M.D., *Shawnee Mission*
Timothy M. Scanlan, M.D., *Salina*

The committee has addressed three primary issues: credentialing, joint educational programs, and problems facing both organizations as a result of changes in federal and state regulations.

The committee is currently working toward developing guidelines for the credentialing process for

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consideration by KHA and KMS Boards, and subsequently for dissemination to the membership of the two organizations. Proposed guidelines would include the construction of a recommended application form and bylaw provisions.

Secondly, the committee will pursue a program of membership education on changes in federal and state regulations covering reimbursement.

Finally, the committee will make specific recommendations on a format by which medical staff and hospital administrators can work jointly on issues related to recent changes in reimbursement and the changing roles and responsibilities of each respective group.

LOCAL HEALTH DEPARTMENTS STUDY

The committee was to investigate several problem areas:

- State financing for local health departments;
- Problems between state and local health departments; and
- Any problems in local health departments.

During the several committee meetings, we heard by letter or by direct contact from physicians in various-sized communities in Kansas, as well as the presidents of public health associations and the Director of the Kansas Department of Health. We arrived at the following conclusions:

- State and local health departments have good working relationships without an undue amount of conflict.
- Local health departments must maintain as much contact as possible, with physicians requested to increase their involvement.
- Primary funding should be local (and thus control stays local) with state funding for mandated programs and for special situations.
- Large health departments appear to be well organized and provide many services with one major problem — the need for additional funding.
- Smaller health departments also meet a great many needs in their communities and in some cases have a special problem — the intrusion of non-physician personnel in medical activities. These problems could be handled by:
 - — increasing physician participation;
 - — resolving issues of conflict with other members of the local health department team or with the county commissioners;
 - — establishing a physician liaison committee with the KMS and the Kansas Department of Health; and
 - — developing protocols and direction by physicians and other personnel.

Members of the committee included:
James A. Loeffler, M.D., Chairman, *Wichita*
Garland L. Campbell, M.D., *Arkansas City*
Barbara P. Lukert, M.D., *Kansas City*
Alex C. Mitchell, M.D., *Lawrence*
John Neuenschwander, M.D., *Hoxie*
Joseph E. Rich, M.D., *Topeka*
Don R. Tillotson, M.D., *Ulysses*
Linda D. Warren, M.D., *Hanover*

LONG TERM CARE COMMITTEE

The purpose of the committee is to identify problems associated with the care of the aged in nursing homes, and to seek solutions to those problems. The committee is charged with educating physicians about the special needs of the aged, and encouraging active physician involvement. The committee also reviews and comments on state and federal regulations affecting nursing homes.

The committee is composed of the following members:
Roger D. Warren, M.D., Chairman, *Hanover*
Anol W. Beahm, M.D., *Great Bend*
James M. Donnell, M.D., *Wichita*
Donald M. Holsinger, M.D., *Pittsburg*
Philip H. Hostetter, M.D., *Manhattan*
Nancy Macy (Ted), *Salina*
Norman G. Marvin, M.D., *Shawnee Mission*
Roy N. Neil, M.D., *Hays*
G. William Nice, M.D., *Topeka*
James K. O'Toole, M.D., *Newton*
Delbert V. Preheim, M.D., *Newton*
Katherine Pyle (Lucien), *Topeka*

The committee addressed the issue of education from two perspectives. The first was the presentation of a scientific session on the "Health and Care of the Aging" at the 1982 Annual Meeting. This was an excellent program attended by more than 100 physicians. In addition, a special issue of the *Journal of the Kansas Medical Society* (July 1982) was devoted to articles on geriatrics.

The committee reviewed the Long Term Care component of the State Health Plan and provided some constructive comments for improvement.

Other activities of the committee included: a call for papers on gerontology to be published in the *Journal of the Kansas Medical Society* on a regular basis; research on developing a film or videotape of the process involved in moving from the hospital to a nursing home; research on education and a stand-

ardized form to assist nursing home aides in communicating vital signs to physicians; and review of a research proposal from the Kansans for Improvement of Nursing Homes. In addition, the committee is on record favoring physician involvement in the teaching of nurses and aides in the area of gerontology.

MEMBERSHIP RECRUITMENT COMMITTEE

In 1982, the House of Delegates referred resolution 82-4 to the Membership Recruitment Committee for study. The resolution directed that state and county membership application procedures be streamlined. Members of the committee included:

Donald D. Goering, M.D., Chairman, *Coldwater*

Warren E. Meyer, M.D., *Wichita*

Joan Sehdev, M.D., *Topeka*

Linda D. Warren, M.D., *Hanover*

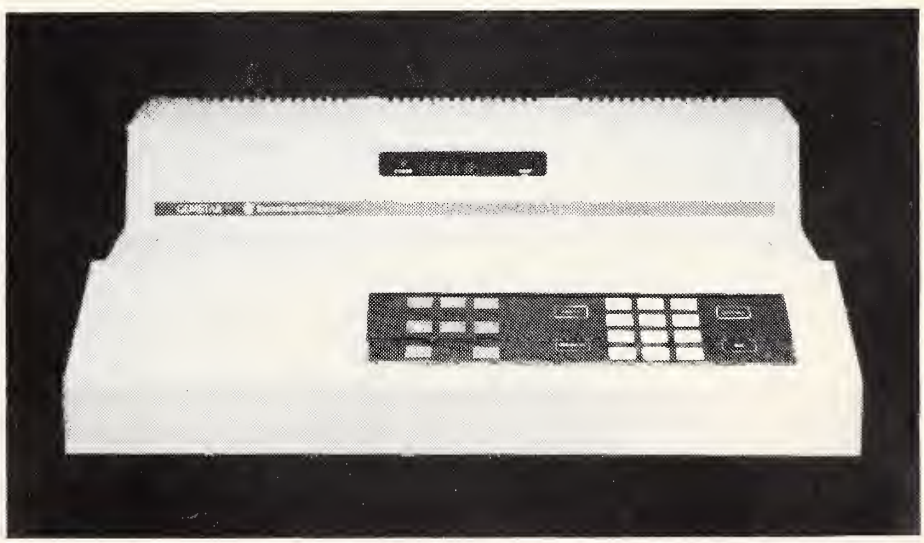
The committee reviewed existing reports and information on membership procedures from the AMA, the American Association of Medical Society Executives, and other states. Initial committee recommendations were to adopt a uniform application form, eliminate waiting periods for membership, reduce probationary membership periods, consider a reciprocity provision for physicians belonging to other medical societies, and to introduce a resolution establishing direct membership provisions for students and residents.

Information on these recommendations and sample forms were mailed to all component medical societies. A meeting of the committee and representatives of Johnson, Sedgwick, Shawnee, and Wyandotte County Medical Societies was held in early March. As a result of this meeting, the committee submits the following recommendations:

- Adopt a uniform membership application form for all component medical societies and the KMS.
- Minimize waiting periods required before accepting members on probationary status.
- Probationary membership should be limited to a maximum of six months.
- There should be no special provisions for reciprocity membership for physicians from other medical societies.
- A resolution will be introduced establishing direct KMS membership provisions for students and

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residents. Sections would be established similar to specialty society sections with delegate representation to the House of Delegates.

STATEWIDE MEDICAL EXAMINER SYSTEM STUDY

The Kansas Medical Society House of Delegates, meeting in Wichita May 2, 1982, created a special committee to study the Medical Examiner system; directed it — through Resolution 82-3 — "to study the concept of a statewide Medical Examiner system" in Kansas; and to report its findings and conclusions to the 1983 House of Delegates.

Members of the committee are:

J. Alan Sanders, M.D., Chairman, *Lawrence*
Cyril W. Black, M.D., *Pratt*
James G. Bridgens, M.D., *Shawnee Mission*
Robert M. Daniels, M.D., *Valley Center*
William G. Eckert, M.D., *Wichita*
L. William Halling, M.D., *Hays*
Ward M. Newcomb, M.D., *Hays*
Claudia M. Pray, M.D., *Leavenworth*
Alex Scott, M.D., *Junction City*

The committee has evaluated the existing medical legal investigative systems of the states of Virginia, Pennsylvania, Oklahoma, Arkansas, Ohio, Province of Ontario, as well as the Manual of the National Association of Medical Examiners. The committee also reviewed Kansas Statutes and the pertinent legislative proposals in 1982 and 1983. The committee further reviewed the Delphi Survey of Kansas Coroners carried out in 1981 by a committee of the Kansas Pathology Society, chaired by Dr. Newcomb, and solicited input from the current district and deputy coroners and from the Kansas Coroners Association.

The committee recommends to the House of Delegates the following observations and findings.

The current coroner system in Kansas is perceived to work well in the metropolitan areas of the state where there is a concentration of physicians, including pathologists, and law enforcement people with ongoing experience in investigating deaths that are possible homicides. In the rural areas, there is indication that the system is significantly less satisfactory. There are problems in finding qualified and motivated physicians to serve as coroners, or deputy coroners, arranging for autopsy services, getting toxicology testing performed in a timely fashion, and having consultative services clearly defined. There is little standardization of investigative procedures for coroners and no review of coroners' performance by someone with recognized competence in this area.

An adequately funded statewide medical examiner system could theoretically address these deficiencies through a central office which would coordinate, educate, and supervise investigations of unusual deaths throughout the state and maintain records and compile statistics. Some committee members expressed concern that such a system would be prohibitively expensive and difficult to administer in Kansas. These concerns need more thorough study with recognition that a medical examiner system might encompass services (such as toxicology) that are currently funded outside the coroner system.

The consensus of the committee is that replacement of the present coroner system with a statewide medical examiner system is not feasible at present but may have potential for future implementation. There is agreement in regard to the following recommendations for improving the present system:

- The present compensation to coroners and deputy coroners should be increased to a level more reasonably in keeping with physicians' compensation for other professional activities.
- The Attorney General must be notified by the District Court of new coroner appointments in order that pertinent information (current laws, etc.) may be forwarded to the newly elected coroner.
- State reporting laws should require hospital administrators to notify District or Deputy Coroners when deaths occur in that hospital.
- The elected law enforcement personnel, particularly in rural counties, need improved education in the area of death investigation.
- All District and Deputy Coroners should be compensated to attend the Western Conference on Criminal and Civil Problems, a semi-annual program of the Milton Helpern International Center of Forensic Sciences offered through Wichita State University.
- The University of Kansas School of Medicine should be encouraged to educate medical students and residents in the area of medical-legal investigation of death as required for review and reporting by the Statutes of the state of Kansas.
- Effort should be made to improve the availability of toxicology expertise and pathology services.
- The coroner jury system of six persons should be changed to a jury of five or seven to avoid deadlock of decisions.
- The Kansas Coroners Association should be sup-

ported and the MANUAL updated to contain pertinent information for coroners.

- A section in *The Journal of the Kansas Medical Society* should regularly be devoted to forensic medicine.

The committee recommends that the Kansas Medical Society House of Delegates support these proposed changes in the present Kansas Coroner System. Additional comments and recommendations of committee members may be attached to the report and distributed to the House of Delegates for information.

WELFARE ADVISORY COMMITTEE

The purpose of the committee is to provide an ongoing liaison with the State Department of Social and Rehabilitation Services (SRS). The committee is composed of the following members:

Ted Young, M.D., Chairman, *Topeka*
Leslie E. Becker, M.D., *Atchison*
Thomas P. Butcher, M.D., *Emporia*
Richard M. Chubb, M.D., *Baxter Springs*
William J. Ciskey, M.D., *Eureka*
Earl F. George, M.D., *Wichita*
Phillip A. Godwin, M.D., *Lawrence*
Charles A. Isaac, M.D., *Newton*
Herman H. Jones, Jr., M.D., *Kansas City*
Francis X. Lenski, M.D., *Iola*
Earl D. Merkel, M.D., *Russell*
Erwin T. Olsen, M.D., *Newton*
David M. Porter, M.D., *Kansas City*
Ben Rubin, Jr., M.D., *Kansas City*
Charles R. Svoboda, M.D., *Chapman*
Danny M. Thompson, M.D., *Kansas City*
Wayne O. Wallace, Jr., M.D., *Atchison*

The committee primarily addresses issues related to the state Medicaid program. KMS members provide medical judgment and make suggestions related to changes in SRS-administered programs. Members of the committee work with SRS to control costs and to reduce hospital admissions to those medically necessary. Efforts have been made to maintain appropriate reimbursement for a list of 139 outpa-

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tient physician procedures and general office visits, contingent upon availability of adequate funds. This level of reimbursement is set at the 75th percentile of the last full year's charges. In addition, the committee has encouraged increases to an adequate level in inpatient fee reimbursement. This level of reimbursement has been frozen for the past eight years at the 50th percentile of 1975 charges. KMS committee members are working toward increases to the 50th percentile of the last full year's charges.

Future projects for the committee will include a review of SRS proposals related to a primary care network and the feasibility of HMOs. In addition, the committee will continue to monitor and have input into changes in the Medicaid program.

WOMEN PHYSICIANS IN ORGANIZED MEDICINE

The KMS Ad Hoc Committee was appointed three years ago and charged with the task of identifying those women, practicing medicine in Kansas, who have leadership qualifications and potential. That goal has been accomplished. Women who are interested and willing to participate in organized medicine have been identified at the state level and their names have been forwarded to the county society level. This past year, the committee was communicating with women in residency programs in Kansas in an attempt to make them aware of the importance of their participation in organized medicine. The response has been good, and this task of encouraging women residents to participate in the KMS both as members and on committees of their choice will be carried on by the KMS Membership Committee. Thus, the groundwork has been laid. A general awareness of the role of women in organized medicine is now apparent. Our task has been completed and we shall, therefore, disband the committee.

There is now apparent, both in Kansas and nationally, an increasing awareness of women physicians in organized medicine. Not only are more women involved actively, but they also have now become aware of an opportunity to participate, which previously they had not even considered.

During these past three years, our efforts have been enhanced in Kansas not only through the support of the KMS Presidents (Drs. Godwin, Hiesterman, and Wedel), but also because of direct communication with the AMA counterpart committee through Dr. Linda Warren, one of five representatives nationwide on the AMA committee.

I have thoroughly enjoyed working not only with the members of this committee, but with all members of the Kansas Medical Society. We in Kansas should be proud to have accomplished our goals ahead of many other states. Serving on this committee were:

Joan Sehdev, M.D., Chairman, *Topeka*
Deloris W. Bell, M.D., *Shawnee Mission*
Phillip A. Godwin, M.D., *Lawrence*
Virginia T. Gruendel, M.D., *Kansas City*
Katherine Pennington, M.D., *Wichita*
Linda D. Warren, M.D., *Hanover*

Note: The following committee reports will be distributed or presented at the annual meeting:

Constitutional Secretary	Legislative
Dispensing Physicians	Mediserve
Editorial Board	Nurse Liaison
Executive Director	Professional Liability
Impaired Physician	Treasurer

TELEPHONE NUMBERS

Lawrence Area Code — 913

Holidome	841-7077
Robinson Gymnasium	864-3371
Alvamar Country Club	842-1907
Lawrence Country Club	843-2938
Cedar Hill Trap Club	843-8213
Elks Club	843-3995

Councilor Reports

Activities in the Council Districts of Kansas

DISTRICT 1

Council District 1 held its annual meeting in Atchison on Wednesday, October 20, 1982. A cocktail hour at 6:30 was followed by a barbecued beef and pork ribs buffet, supplied by Schuetz Locker of Atchison. KMS President Kermit Wedel, M.D., and Steve Carter, KMS Executive Director, held an informal session following the meal to inform those present of the actions, plans, and problems facing the Kansas Medical Society.

WAYNE O. WALLACE, JR., M.D., *Councilor*

DISTRICT 2

Wyandotte County Medical Society (WCMS), which comprises KMS Council District 2, continues to gain in total membership after several years of no activity or a loss in total membership. We increased from a total of 443 members in December 1981 to 485 in December 1982.

With general economic conditions affecting the financial status of the Medical Society, our Budget and Finance Committee was considerably more active this last year. Through their diligent efforts, the financial condition of the Medical Society improved dramatically.

Our Emergency Medical Care Committee also continued to be very active throughout the year. A new Medical Director for K.A.R.E. (our Type I ambulance service run by the Fire Department) was appointed effective January 1, 1983, and the committee has continued to function at its high level of productivity.

The WCMS was involved in discussions and negotiations regarding review programs during the year. The Society decided not to set up its own review process for Medicaid review but instead supported the review function of the Kansas Foundation for Medical Care. This was accepted by the Department of Social and Rehabilitation Services (SRS).

Additionally, we were involved in discussions regarding "private-side" review in the Greater Kansas City area and eventually endorsed a program started by the Midlands Medical Review Program. We appointed a WCMS member to their Board of Directors and also appointed a WCMS member to the Board of Directors of Medical Systems, Inc., a corporation started by the Jackson County Medical Society to do professional review. Both of these organizations contract with the Kansas Foundation for Medical Care to do the actual review of Kansas cases.

Our immediate past president, David Pugh, M.D., was elected to serve as Chairman of the Area Medical Council for 1983. This is an association of all the medical societies, some of the osteopathic societies, and some of the specialty societies located in the Greater Kansas City area.

We were disappointed with our membership turnout, but very happy with KMS President Dr. Kermit Wedel's appearance before the Wyandotte County Medical Society on October 19, 1982. This was also the first opportunity some of our members had to meet Steve Carter, Executive Director.

LOUIS M. CULP, M.D., *Councilor*

DISTRICT 3

The Johnson County Medical Society continues to grow and expand its activities. Fourteen new active members were added during 1982, bringing the active membership to 267. In addition, one corresponding member has been added.

Monthly meetings continue to be well attended, with programs of significant interest to all members. Program topics during the past year include intervention radiology, cardiac exercise, Chinese medical care, legislative topics, and risk management. In October 1982, Kermit G. Wedel, M.D., KMS President, visited the society. He discussed the broad activities of KMS in relationship to the ever changing world in which we practice.

The society is participating in a physician referral program jointly with Clay and Jackson County (Missouri) Medical Societies. A large display advertisement has been placed in the Yellow Pages, offering the services of the three county societies in referring patients to members. The results of this advertisement have been quite gratifying, as numerous requests are received by each of the societies.

JAMES G. BRIDGENS, M.D., *Councilor*

DISTRICT 4

Council District 4 welcomed KMS President Kermit G. Wedel, M.D., on January 25, 1983, with a

well attended lively discussion.

GEORGE W. POGSON, M.D., *Councilor*

DISTRICT 6

Our active year begins with new officers assuming their duties in September. President Robert Durst, M.D., opened our first meeting in September 1982 on the new West Campus of the Menninger Foundation. Shawnee County Medical Society (SCMS) members were guests of the Foundation and toured the new facilities.

SCMS gained a total of seven members in 1982, bringing total membership to 342. Of these, 285 are active, 46 have emeritus or retired status, and the others are residents, associates, or are in other categories. One of our members, Floyd Beelman, M.D., was honored by the Health Department for his many years of service, and an article about his career was published on the front page of the *Topeka Capital Journal*. Also in 1982, long time Topeka physician and KMS member Howard U. Kennedy died suddenly.

The SCMS Public Relations Committee and the Board of Directors of the Society developed a "Guide to Health Services in Shawnee County" which was published in 1982 in an attractive format and distributed free of charge to numerous individuals and organizations throughout Shawnee County. The guide is not a physician directory. It does not take the place of the SCMS listing in the Yellow Pages of the telephone book, for example. Rather, it is designed to help people identify sources of regular and emergency care in advance of need. The booklet includes chapters on "How to select a doctor for your family"; "Medical services"; "What is the medical society?"; "Medical and surgical specialties and what they do"; "Topeka hospitals"; and chapters listing all health care and service organizations in the community as well as nursing homes that are available. Emergency services have their own section with easy-to-read telephone numbers for poison control centers, fire, police, ambulance, and other services. SCMS would be happy to provide other county societies with a sample of our guide if they are interested in developing a similar publication for local distribution.

The society also made a substantial donation to the Stormont-Vail Education Center currently nearing completion. It will be an outstanding education asset for our region.

We continue to publish a bi-monthly bulletin for our members. The bulletin summarizes minutes of meetings, publishes pictures and vital statistics on new probationary members to help familiarize the rest of the medical community with them and their specialties, and publishes useful information of current concern to the membership, such as advice on "Cutting the Physician/Patient Relationship" and "The FTC: questions and answers."

JAMES H. RANSOM, M.D., *Councilor*

TELEPHONE NUMBERS

Lawrence Area Code — 913

Holidome	841-7077
Robinson Gymnasium	864-3371
Alvamar Country Club	842-1907
Lawrence Country Club	843-2938
Cedar Hill Trap Club	843-8213
Elks Club	843-3995

DISTRICT 7

Council District 7, nearly synonomous with the Flint Hills District Medical Society (FHDMS), meets monthly except during the summer, usually at the Emporia Country Club or the American Legion in Emporia. Richard Frazier, M.D., passed the reins to Dave Edwards, M.D., in January.

Highlight of the year for this area was the installation of the latest, most up-to-date CT scanner available, privately donated to St. Mary's Hospital, and presently in use by our three radiologists; they expect Mark Witaczack, M.D., now in residency at the Cleveland Clinic, to join their ranks in July. In a cooperative venture between the Emporia hospitals, with the CT scanner at St. Mary's Hospital, digital

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angiography will be installed at Newman Memorial Hospital. This equipment will provide less invasive techniques for studying carotid arteries and other arterial vascular structures. Newman Hospital has also purchased a new nuclear medicine camera for tomographic evaluation of structures and to enhance cardiac thallium studies.

Some of our scientific programs for the FHDMS have helped to educate us in the proper use of this new equipment. John A. Segerson, M.D., neurologist from Topeka's Menninger Clinic, discussed the use of the CT scanner and the evaluation of the stroke patient. William J. Walls, M.D., of Topeka, presented a program on the use of ultrasound and CT in examination of the abdomen.

Jim Glenn, M.D., local orthopedic surgeon, presented a program on the dynamics and anatomy of low back pain. Perri Ginder, M.D., presented a program on new modalities of treatment of rheumatoid arthritis.

James Bergin, M.D., Chief of Internal Medicine of Bethany Hospital and a clinical professor of medicine at the University of Kansas School of Medicine, returned this year to give us another outstanding slide presentation.

The FHDMS supported Gridley, Kansas', application as a medically underserved area.

Physicians who practice internal medicine, family practice, and gynecology and obstetrics have all had students from UKSM-KC spend time with them.

A request has been received from a medical student from the University of Dusseldorf in West Germany to spend eight weeks in a clinical rotation with local physicians at Newman Hospital.

With the recent addition of new diagnostic modalities, physicians in this district are better able to serve their patients. Nearly all physicians in Council District 7 are members of the Flint Hills District Medical Society. New physicians already in practice are internist James Barnett, M.D., from Parkland Hospital in Dallas; surgeon Ron Farris, M.D., from St. Luke's Hospital and Truman Medical Center in Kansas City, Missouri; psychiatrist Marucio Masferrer, M.D., from the Menninger Clinic and recently returned from Canada; family physician Chuck Allred, M.D., practicing at Council Grove; and dermatologist Hugh Bailey, M.D., from Kansas City, practicing in the Flint Hills Medical Building one day per week. Family physician Mark Pearson, M.D., from Carbondale, Illinois, is planning to establish practice in the district. John Best, M.D., will leave Emporia for a fellowship in cardiology at UKSM. The FHDMS is still seeking a full-time resident ENT specialist.

Many members of the FHDMS have been active in their own specialty societies as well as such organizations as the local chapters of the American Heart Association and the American Cancer Society.

It has been a pleasure to associate with councilors, officers, and staff of the KMS while representing the FHDMS.

MARVIN D. SNOWBARGER, M.D., *Councilor*

DISTRICT 8

KMS President Dr. Kermit Wedel visited the District 8 meeting on October 21, 1982. Dr. Wedel brought us up to date on the activities of the State Society. A personal invitation was sent to members of the Butler-Greenwood Society, but their attendance was limited.

There seems to be very little dialogue between the county societies in this council district. If organized medicine is to survive, there needs to be more cooperation between the various societies and their members.

Cowley County Medical Society has been having interesting meetings on the third Thursday of each month with a program speaker from the faculty of the University of Kansas School of Medicine-Wichita. This seems to have stimulated attendance. Those attending receive one hour of continuing medical education credit. We would welcome any physician from the Butler-Greenwood Society.

Many of the Cowley County members are looking forward to the State Meeting in May in Lawrence. The Society has not met there before, and I am certain that the Douglas County group will have a super program.

NEWTON C. SMITH, M.D., *Councilor*

DISTRICT 9

The medical climate in District 9 has been stable. There have been many new physicians who have entered practice, and others who have retired from practice.

President Kermit G. Wedel, M.D., attended our district meeting at Salina, outlining problems and solutions of the health care delivery system in the state of Kansas.

It appears that the biggest problem confronting the physicians of Kansas is reimbursement policies from Medicare and Medicaid sources, as well as federal and state rules and regulations.

H. D. DOUBEK, M.D., *Councilor*

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MEPROBAMATE: DRUG DEPENDENCE: Physical and psychological dependence, and abuse have occurred. Chronic intoxication from prolonged ingestion of, usually, greater than recommended doses is manifested by ataxia, slurred speech, and vertigo. Therefore, carefully supervise dose and amounts prescribed and avoid prolonged use, especially in alcoholics and others with known propensity for taking excessive quantities of drugs. Sudden withdrawal after prolonged and excessive use may precipitate recurrence of preexisting symptoms, e.g. anxiety, anorexia, or insomnia, or withdrawal reactions, e.g., vomiting, ataxia, tremors, muscle twitching, confusional states, hallucinosis, and, rarely, convulsive seizures. Such seizures are more likely in persons with CNS damage or preexistent or latent convulsive disorders. Onset of withdrawal symptoms occurs usually within 12 to 48 hours after discontinuation; symptoms usually cease within next 12-to-48-hour period. When excessive dosage has continued for weeks or months, reduce dosage gradually over 1 to 2 weeks rather than stop abruptly. Alternatively, a short-acting barbiturate may be substituted, then gradually withdrawn.

POTENTIALLY HAZARDOUS TASKS: Warn patients meprobamate may impair mental or physical abilities required for potentially hazardous tasks, e.g., driving or operating machinery.

ADDITIVE EFFECTS: Since CNS-suppressant effects of meprobamate and alcohol or meprobamate and other psychotropic drugs may be additive, exercise caution with patients taking more than one of these agents simultaneously.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during first trimester of pregnancy, has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at time of institution of therapy should be considered. Advise patients if they become pregnant during therapy or intend to become pregnant to communicate with their physicians about desirability of discontinuing the drug.

Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breastfeeding patients, consider the drug's higher concentrations in breast milk as compared to maternal plasma levels.

USAGE IN CHILDREN: Keep preparations with aspirin out of reach of children. Equagesic®-M is not recommended for patients 12 years of age and under.

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MEPROBAMATE: Use lowest effective dose, particularly in elderly and/or debilitated, to preclude over-sedation. Meprobamate is metabolized in the liver and excreted by the kidney; to avoid excess accumulation exercise caution in its use in patients with compromised liver or kidney function. Meprobamate occasionally may precipitate seizures in epileptic patients. It should be prescribed cautiously and in small quantities to patients with suicidal tendencies.

ADVERSE REACTIONS: ASPIRIN: May cause epigastric discomfort, nausea, and vomiting. Hypersensitivity reactions, including urticaria, angioneurotic edema, purpura, asthma, and anaphylaxis may rarely occur. Patients receiving large doses of salicylates may develop tinnitus.

MEPROBAMATE: CNS: Drowsiness, ataxia, dizziness, slurred speech, headache, vertigo, weakness, paresthesias, impairment of visual accommodation, euphoria, overstimulation, paradoxical excitement, fast EEG activity.

GI: Nausea, vomiting, diarrhea.

CARDIOVASCULAR: Palpitation, tachycardia, various forms of arrhythmia, transient ECG changes, syncope, hypotensive crisis.

ALLERGIC OR IDIOSYNCRATIC: Milder reactions are characterized by itchy, urticarial, or erythematous maculopapular rash, generalized or confined to the groin. Other reactions include leukopenia, acute nonthrombocytopenic purpura, petechiae, ecchymoses, eosinophilia, peripheral edema, adenopathy, fever, fixed drug eruption with cross-reaction to carisoprodol, and cross-sensitivity between meprobamate/mebutamate and meprobamate/carbromal. Rare, more severe hypersensitivity reactions include hyperpyrexia, chills, angioneurotic edema, bronchospasm, oliguria, and anuria. Also, anaphylaxis, exfoliative dermatitis, stomatitis, and proctitis. Stevens-Johnson syndrome and bullous dermatitis have occurred.

HEMATOLOGIC (SEE ALSO "ALLERGIC OR IDIOSYNCRATIC"): Agranulocytosis, aplastic anemia have been reported, although no causal relationship has been established, and thrombocytopenic purpura.

OTHER: Exacerbation of porphyric symptoms.

DOSAGE AND ADMINISTRATION: Usual dose is one or two tablets, 3 to 4 times daily as needed for relief of pain when tension or anxiety is present. Not recommended for patients 12 years of age and under.

OVERDOSAGE: Treatment is essentially symptomatic and supportive. Any drug remaining in the stomach should be removed. Induction of vomiting or gastric lavage may be indicated. Activated charcoal may reduce absorption of both aspirin and meprobamate. Aspirin overdosage produces usual symptoms and signs of salicylate intoxication. Observation and treatment should include management of hyperthermia, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions. Suicidal attempts with meprobamate have resulted in drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse. Some suicidal attempts have been fatal. The following data, reported in the literature and from other sources, are not expected to correlate with each case (considering factors such as individual susceptibility and length of time from ingestion to treatment), but represent usual ranges reported. Acute simple overdose (meprobamate alone): Death has been reported with ingestion of as little as 12 gram meprobamate and survival with as much as 40 gram.

BLOOD LEVELS:

0.5-2.0 mg percent represents usual blood-level range after therapeutic doses. The level may occasionally be as high as 3.0 mg percent.

3-10 mg percent usually corresponds to findings of mild-to-moderate symptoms of overdosage, such as stupor or light coma.

10-20 mg percent usually corresponds to deeper coma, requiring more intensive treatment. Some fatalities occur.

At levels greater than 20 mg percent, more fatalities than survivals can be expected.

Acute combined overdose (meprobamate with other psychotropic drugs or alcohol): Since effects can be additive, history of ingestion of a low dose of meprobamate plus any of these compounds (or of a relatively low blood or tissue level) cannot be used as a prognostic indicator.

In cases of excessive doses, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Any drug remaining in stomach should be removed and symptomatic treatment given. Should respiration or blood pressure become compromised, respiratory assistance, CNS stimulants, and pressor agents should be administered cautiously as indicated. Diuresis, osmotic (mannitol) diuresis, peritoneal dialysis, and hemodialysis have been used successfully in removing both aspirin and meprobamate. Alkalinization of the urine increases excretion of salicylates. Careful monitoring of urinary output is necessary, and caution should be taken to avoid overhydration.

Relapse and death, after initial recovery, have been attributed to incomplete gastric emptying and delayed absorption.

HOW SUPPLIED: Bottles of 50 scored tablets.

℗ 1983, Wyeth Laboratories

Wyeth Laboratories
Philadelphia, Pa 19101

DISTRICT 10

District 10 physicians met with KMS President Kermit Wedel, M.D., and Mrs. Wedel at Halstead on November 23, 1982. Also in attendance were Steve Carter, Executive Director, and Roger Warren, M.D., who represented KaMPAC.

Douglas Voth, M.D., of the University of Kansas School of Medicine-Wichita, spoke briefly regarding problems facing the medical school.

Richard Siemens, M.D., Lyons, was elected Councilor for District 10 to succeed the present councilor, who was ineligible for re-election. Varden J. Loganbill, M.D., Moundridge, was elected alternate councilor.

Dr. Wedel gave us an overview of the political and economic changes on the horizon for medicine. A spirited discussion followed Dr. Wedel's talk.

HERBERT FRANSEN, M.D., *Councilor*

DISTRICT 11

The Medical Society of Sedgwick County continues to be active during 1982-83 under the leadership of Drs. W. G. Cauble and Terry Poling. The Society's current membership, including all categories, is 733. Of this number, 601 are actively practicing medicine and 54 are residents and students; the remaining 78 are retired or exempt. The Society's executive office staff consists of six full time employees.

During the year the Society has become involved in many programs; the principal ones are reviewed here.

Foundation Review Program

The Foundation program continues to expand. It was established in 1981 to assure the public, the insurance industry, and management that in-hospital admission and continued stay of patients were based on medical necessity. The program covers approximately 52,000 citizens of Sedgwick County. The program began with the Boeing Company, and Beech Aircraft Company was added in April 1982. The Medicaid population in Sedgwick County was added in September 1982, and union employees of the Vulcan Chemical Company in January 1983. This program continues to operate smoothly. Utilizing reports from company management and the involved insurance companies, the program has been instrumental in modifying practice patterns of physicians and has been cost effective. Several other business groups in Wichita and Sedgwick County have indicated an interest in joining the review program. It is anticipated that further expansion will take place in 1983. The Foundation review program now employs six persons in addition to the direct assistance provided by the Society staff and physician members.

Program for Unemployed Citizens

Due to the large number of layoffs in the Wichita community, the Society was approached by various community groups concerning the problems faced by unemployed citizens in obtaining needed medical care. The Society, in cooperation with local hospitals and the Osteopathic Society, agreed to organize and implement a program whereby needed (non-elective) medical care would be made available to this group of citizens at reduced costs in accordance with established guidelines. The program was initiated in early January with approximately 350 members of the Society agreeing to participate. A local laboratory has agreed to provide all needed laboratory services with some of the local pharmacists providing required medications on a cost basis. The hospitals are providing services with charges based on the family's financial situation. As of February 16, 350 families have applied for assistance. Based

TELEPHONE NUMBERS

Lawrence Area Code — 913

Holidome	841-7077
Robinson Gymnasium	864-3371
Alvamar Country Club	842-1907
Lawrence Country Club	843-2938
Cedar Hill Trap Club	843-8213
Elks Club	843-3995

on comments from citizens, physicians and hospitals, the program is being well received and is operating very effectively.

Medical Careers Loan Fund

In view of cut-backs at both state and federal levels in financial assistance to medical students, the Society's loan fund has observed an increase in applications and it appears that the number of applications will increase considerably during 1983. Since 1960 when the program was established, 53 students have received financial assistance. Of this number, 45 have been medical students. Through the program the student is provided a maximum annual loan of $4,000. Payment is made directly to the involved school. Loans are to be repaid over a period of time at a low rate of interest. Operating funds are derived from contributions from the Society's auxiliary, individual physicians, and the Society itself as well as through repayment of loans. Of students who have received assistance, 19 have repaid their loans in full. Collection problems have arisen in only one situation.

Community Cost Containment

In October 1982, the Society, in cooperation with representatives of the hospital, business and insurance community, conducted its fourth informational seminar on health care costs and cost containment. This program continues to be well received by the business community. The Society was one of the principal organizers of this program and consideration is being given to expanding the organizational structure to include additional business and community representatives.

Paging System

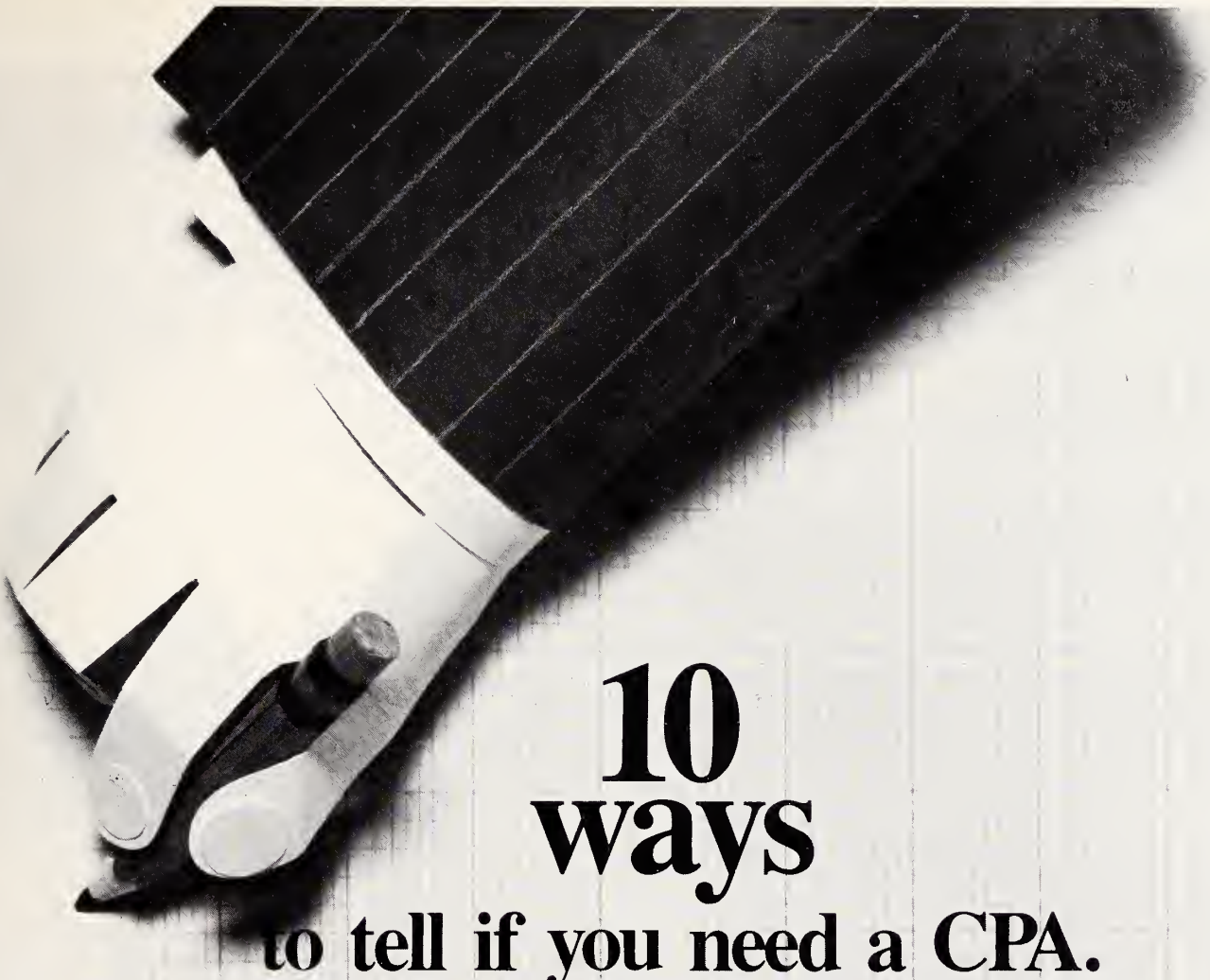
The Society's paging system, which was establishing in 1972, continues to provide a needed communication service to both the physician and hospital. The system, now serving approximately 500 local physicians or their employees, provides a mechanism whereby a physician may be paged and given a voice message from any phone in the Wichita hospitals and Physician Exchange. Although providing a needed service, the ever occurring beep-beep can become quite annoying.

The Kansas Foundation for Medical Care, Inc. (KFMC), Topeka, is seeking to fill the half-time position of Medical Director of KFMC.

To obtain a Position Prospectus, address a written request to:

SELECTION COMMITTEE
KANSAS FOUNDATION FOR MEDICAL CARE
1000 WEST TENTH
TOPEKA, KANSAS 66604

All requests will be handled in a confidential manner.



10 ways to tell if you need a CPA.

- ☐ **1** Are your tax returns getting complicated? (Perhaps because family earnings are now from *several* sources or because your business is growing or changing?)
- ☐ **2** Do you need *immediate* tax write-offs through proper tax planning?
- ☐ **3** Would you consider changing some business procedures to cut *future* taxes?
- ☐ **4** Are you setting up or changing estate plans for yourself or someone else?
- ☐ **5** Is your business required to file financial reports — anything from a statement of earnings to full-scale audited financial statements?
- ☐ **6** Do you feel it might be time to incorporate your business?
- ☐ **7** Are you preparing a business loan application — and need supporting documentation?
- ☐ **8** Do you want to set up or improve a bookkeeping system?
- ☐ **9** Are you thinking of starting a pension or profit-sharing plan?
- ☐ **10** Do you need an *independent* source of professional management advice on how to use your resources more efficiently or how to make financial projections?

If you answered "yes" to any question, seek out a certified public accountant. You'll be dealing with a professional who has the education and experience to handle *all* the above situations — and many more.

There's generally no charge for the initial consultation. And, if you're not yet ready to use these services *advantageously,* a CPA will tell you. So, you've nothing to lose; much to gain. Go ahead, make that important call.

KS CPA

Kansas Society of Certified Public Accountants

Membership Involvement

During the year, extra effort has been made to involve more members in Society activities through bylaw amendments. The number of members serving on various commissions and committees has been increased in order to obtain the views and impact of a large cross section of the Society. During the year, approximately 220 Society members were involved to some degree in Society activities.

Student and Resident Members

Extra effort has been made throughout the year to bring medical students and residents into Society membership. Although the numbers have increased somewhat, these activities will be expanded during the remainder of 1983 through more direct contact with this group of physicians.

In summary, this past year has been a busy and satisfying year for the MSSC. We look forward to addressing the challenging problems that lie ahead.

K. William Bruner, Jr., M.D., *Councilor*

DISTRICT 12

Council District 12 is composed of the Ninnescah County Medical Society (Pratt, Kingman, and Stafford counties) and the South Central Tri County Medical Society (Barber, Harper, and Sumner counties).

Carl Ambler, M.D., Pratt, has been appointed new District 12 councilor from the Ninnescah Society, and Joel Wiegand, M.D., Wellington, has been selected by the Tri County Society to serve as alternate councilor.

The Pratt Hospital, with its $7 million modification and improvement, is a facility that is well utilized. A new internist, Steve Dillon, M.D., has established practice in Pratt. Dr. Warren Jacks is semi-retired and has moved to Arkansas. Dr. Chaffin has relocated in Tennessee, and Dr. Thomas has relocated in Oklahoma. Dr. Thorpe from the Ninnescah Society has retired.

In the Wellington area of District 12, progress is seen in a new ICU with nuclear medicine and sonography capabilities at St. Lukes Hospital. This area is newly served by an orthopedist, a cardiologist, and a urologist on an intermittent basis.

During 1982, a newly remodeled clinic in Anthony made space available for one of the three new physicians now practicing in this area.

P. J. Antrim, M.D. *Councilor*

DISTRICT 13

Central Kansas Medical Society now includes 74 physicians. Officers for 1983 are Dallas Richards, M.D., President; Robert Enberg, M.D., Vice President; and Howard Wilcox, M.D., Secretary/Treasurer. New members include Dr. Rick Kellerman, Plainville, and Drs. John Dorsch and A. Christine Kelly, Hays.

We are saddened to report that Drs. Peter K. Wiens and Frank A. Dlabal have been added to our list of decreased members. Dr. Anibal Medina has left the local medical community to practice his specialty of urology with the United States Air Force.

Wallace N. Weber, M.D., *Councilor*

DISTRICT 15

The meeting of District 15 was held at the Liberal Country Club on October 28, 1982. KMS President Kermit Wedel, M.D., talked on developments in the Legislature of interest to the Society, and Steve Carter, Executive Director, attended and met mem-

(Continued on page 221)

TELEPHONE NUMBERS

Lawrence Area Code — 913

Holidome	841-7077
Robinson Gymnasium	864-3371
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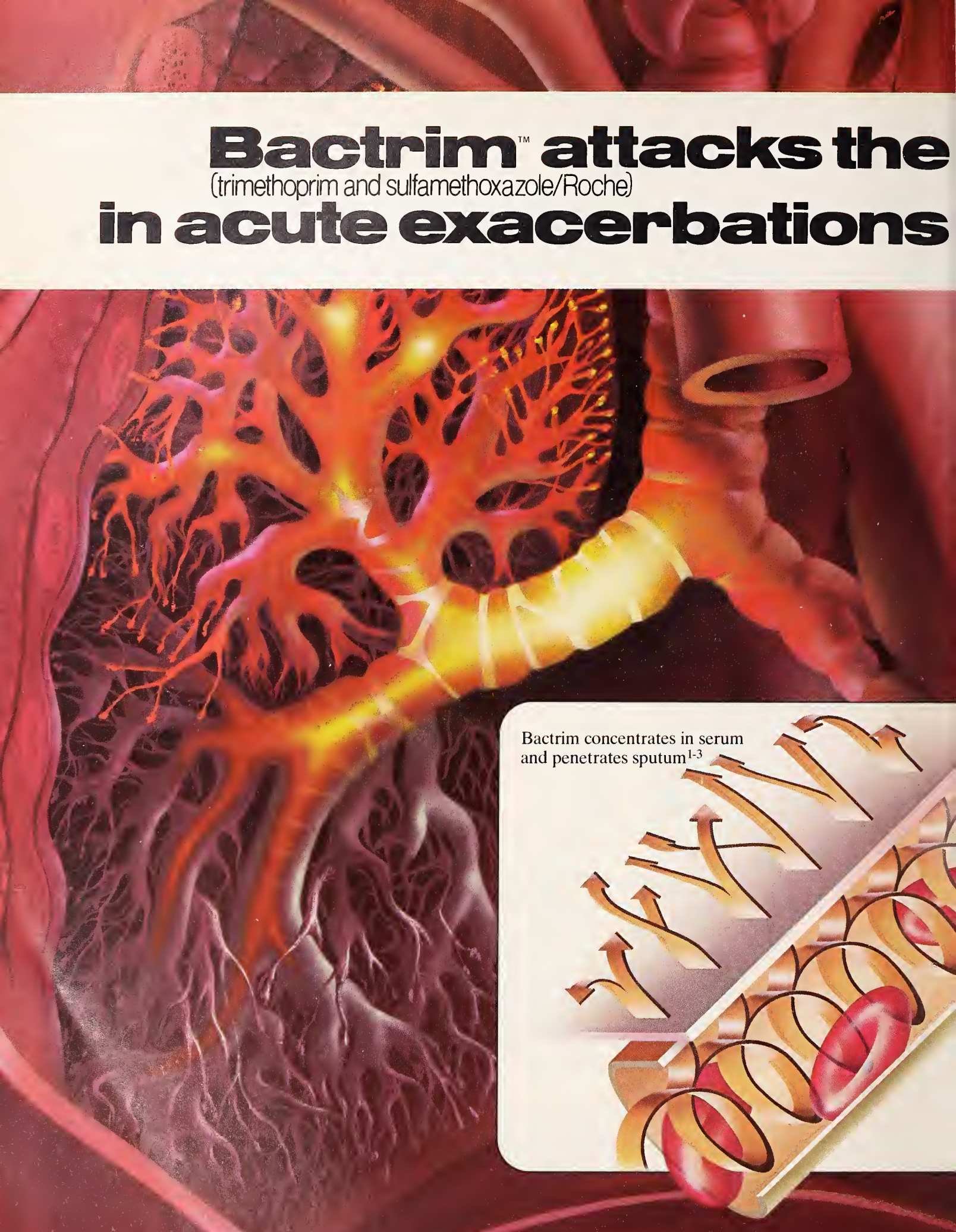
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J-9043-4 July 1984

Bactrim™ attacks the
(trimethoprim and sulfamethoxazole/Roche)
in acute exacerbations



Bactrim concentrates in serum and penetrates sputum[1-3]

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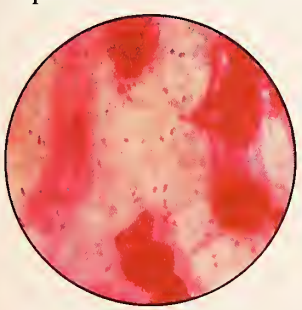
major pathogens of chronic bronchitis*

Bactrim clears sputum of susceptible bacteria

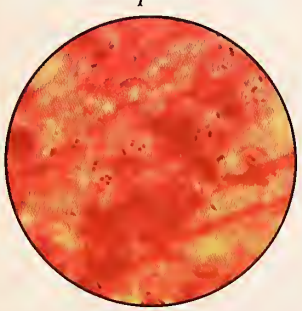
In sputum cultures from patients with acute exacerbations of chronic bronchitis, *H. influenzae* and *S. pneumoniae* are isolated more often than any other pathogens.[4,5] One study of transtracheal aspirates from 76 patients with acute exacerbations found that 80% of the isolates were of these two pathogens.[5]

Bactrim is effective *in vitro* against most strains of both *S. pneumoniae* and *H. influenzae*—even ampicillin-resistant strains. And in acute exacerbations of chronic bronchitis involving these two pathogens, sputum cultures taken seven days after a two-week course of therapy showed that Bactrim eradicated these bacteria in 91% (50 of 55) of the patients treated.[6]

attacks *H. influenzae*—even ampicillin-resistant strains



attacks *S. pneumoniae*



Bactrim reduces coughing and sputum production

In three double-blind comparisons with ampicillin *q.i.d.*, Bactrim DS proved equally effective on all clinical parameters.[7-9] Bactrim reduced the frequency and severity of coughing, reduced the amount of sputum produced and cleared the sputum of purulence.

Bactrim has the added advantages of *b.i.d.* dosage convenience and a lower incidence of diarrhea than with ampicillin, and it is useful in patients allergic to penicillins.

Bactrim also proved more effective than tetracyclines in 10 clinical trials involving nearly 700 patients.[10] Overall clinical condition of the patients, changes in sputum purulence, reduction in sputum volume and microbiological clearance of pathogens—all improved more with Bactrim therapy than with tetracyclines. G.I. side effects occurred in only 7% of patients treated with Bactrim compared with 12% of tetracycline-treated patients. (See Adverse Reactions in summary of product information on next page.)

Bactrim is contraindicated in pregnancy at term and nursing mothers, infants under two months of age, documented megaloblastic anemia due to folate deficiency and hypersensitivity.

Bactrim DS. For acute exacerbations of chronic bronchitis in adults* when it offers an advantage over single-agent antibacterials.

References: **1.** Hughes DTD, Bye A, Hodder P: *Adv Antimicrob Antineoplastic Chemother I*/2:1105-1106, 1971. **2.** Jordan GW *et al: Can Med Assoc J 112*:91S-95S, Jun 14, 1975. **3.** Beck H, Pechere JC: *Prog Antimicrob Anticancer Chemother 1*:663-667, 1969. **4.** Quintiliani R: Microbiological and therapeutic considerations in exacerbations of chronic bronchitis, in *Chronic Bronchitis and Its Acute Exacerbations: Current Diagnostic and Therapeutic Concepts;* Princeton Junction, NJ, Communications Media for Education, Inc., 1980, pp. 9-12. **5.** Schreiner A *et al: Infection 6*(2):54-56, 1978. **6.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **7.** Chodosh S: Treatment of acute exacerbations of chronic bronchitis: results of a double-blind crossover clinical trial, in *Chronic Bronchitis and Its Acute Exacerbations: Current Diagnostic and Therapeutic Concepts. Op. cit.*, pp. 15-16. **8.** Chervinsky P: Double-blind clinical comparisons between trimethoprim-sulfamethoxazole (Bactrim™) and ampicillin in the treatment of bronchitic exacerbations. *Ibid.*, pp. 17-18. **9.** Dulfano MJ: Trimethoprim-sulfamethoxazole vs. ampicillin in the treatment of exacerbations of chronic bronchitis. *Ibid.*, pp. 19-20. **10.** Medici TC: Trimethoprim-sulfamethoxazole (Bactrim™) in treating acute exacerbations of chronic bronchitis: summary of European clinical experience. *Ibid.*, pp. 13-14.

Economical b.i.d.

Bactrim™ DS

(160 mg trimethoprim and 800 mg sulfamethoxazole/Roche)

*Due to susceptible organisms. **Please see next page for summary of product information.**

Bactrim™

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:

Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS:

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100; Prescription Paks of 20 and 28. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. *Pediatric Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz (1 pint). *Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).

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Councilor Reports

(Continued from page 220)

bers of the district, many of them for the first time.

A number of new members from Dodge City were present at the meeting, and several prospective members also attended.

EDWIN D. RATHBUN, M.D., *Councilor*

DISTRICT 16

Northwest Kansas Medical Society continues to hold its meetings in conjunction with the Kansas Circuit Course for Physicians offered by the University of Kansas School of Medicine six times each year.

We welcomed three new members this past year: Rex Kolste, M.D., Colby; Jack Meyer, M.D., Norton; and George Erbacher, M.D., Atwood. We have lost one member as Dr. George Marshall of Colby has retired and moved out of state.

We welcomed the President of KMS, Kermit Wedel, M.D., as guest of honor at our meeting on October 6, 1982. We wish to thank him and all those accompanying him for their time and efforts in keeping us up to date on medicine in Kansas.

JOHN R. NEUENSCHWANDER, M.D., *Councilor*

DISTRICT 17

John Gilbert, M.D., has provided excellent leadership for the Southwest Kansas Medical Society during 1982. The inclusion of the Auxiliary, guest speakers, and scientific programs have significantly improved interest and attendance at our quarterly meetings.

The May meeting included the film "Patton, M.D." and a lively discussion; a symposium on retirement was presented at the September meeting. Kermit G. Wedel, M.D., KMS President, spoke to us at our November meeting, as did Charles Hartman, M.D., from the University of Kansas School of Medicine, and Steve Carter, our Executive Director. Kathy Wedel provided enthusiasm and encouragement to Vi Arroyo and Phyllis Bigler on Auxiliary matters. Our January scientific session was on the "Toxic Shock Syndrome," presented by Dak Burnett, M.D., surgery resident from UKSM. New officers for 1983 are Luther Fry, M.D., President; Frank Hansen, M.D., Vice President; and Frank Eichhorn, M.D., Secretary-Treasurer.

We welcome physicians new to our area since our last report, and express our continued thanks to the many residents and medical students who have kept us excited about the newness of medicine and their careers in future medical-surgical practices.

Our continued thanks also to the many physicians who have given anonymous support to the Impaired Physician Program this past year. We all look forward to 1983 with its problems and opportunities.

MAX E. TEARE, M.D., *Councilor*

DISTRICT 18

Activities in our district have varied through the year. Garnett has acquired two new physicians — David Henderson, M.D., and Roger Victorina, M.D. — both in Family Practice.

Steve Saylor, M.D., formerly of Ottawa, has transferred his Family Practice to Topeka. The Jaycee Janes in Ottawa have worked diligently to furnish and equip a pediatric room at the Ottawa Hospital. Their efforts are greatly appreciated.

Lawrence has gained three new physicians this year: Lisa Campbell, M.D., Pediatrics; Rich Sozinski, M.D., Internal Medicine; and Lyda Osborn, M.D., Pulmonary Specialist.

The Douglas County Medical Society had the pleasure of hosting the district meeting for the visiting KMS President, Kermit Wedel, M.D. He was able to answer many questions that concern our members. We will also have the pleasure of hosting the Kansas Medical Society meeting this spring. Many hours of planning have gone into this meeting. There will be some variation in the format, and we hope that all who attend will enjoy the changes. The professional program will cover the field of cancer, and many excellent speakers have been invited to participate.

The latest development is the thrust in our direction of the pre-paid medical plans, which are a derivative of the HMO system. The IPA, which is sponsored by Blue Cross-Blue Shield, has met with the Medical Society here in Lawrence and given us all the information about their plans. They propose to offer contracts later this year. Their plan will include any physician who wishes to classify him/herself as a primary care provider. An HMO representative from Wichita visited, but was more secretive about their operation. They are, apparently, interested only in groups. This method of medical care financing, of course, is new to all concerned. The area will have to wait and see all that occurs with these two plans.

ROBERT W. HUGHES, M.D., *Councilor*

DISTRICT 19

The Southeast Kansas Medical Society continues to hold monthly meetings in Independence. Associated with these meetings are continuing medical education courses offered through the University of Kansas School of Medicine, although the physician who helped to organize the courses has since moved out of the district.

During the last year there has been a net loss of ten active members in this district. While three new members have joined, this was more than offset by those who moved, died, or simply dropped from the Society rolls. At one point during the year, Albert Kihm, M.D., was forced to serve as president, vice-president, and secretary of our society. Fortunately, Ed Robinson, M.D., as he has for many years, has continued to serve as treasurer.

In addition to speakers from UKSM, our society was privileged to be visited by KMS President Kermit Wedel, M.D., and Steve Carter, Executive Director.

Robert J. Haskins, M.D., *Councilor*

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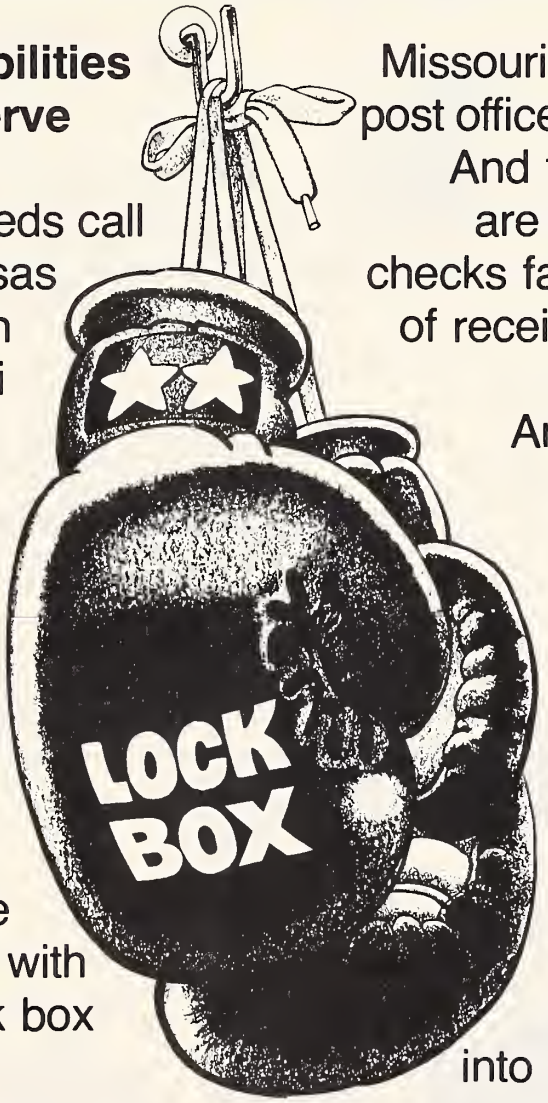
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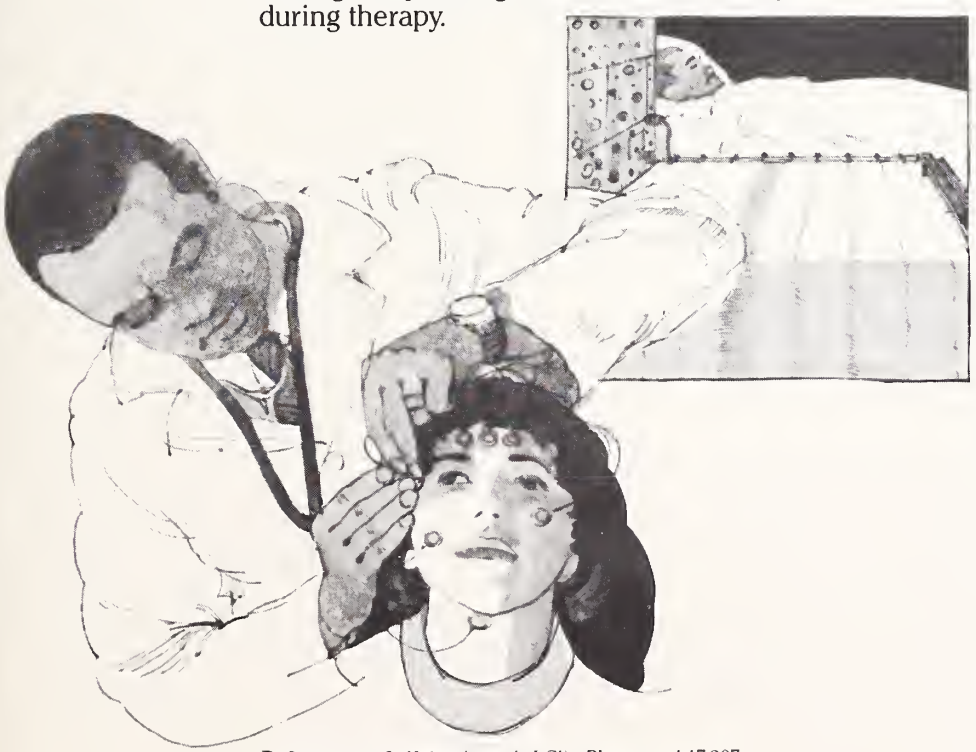
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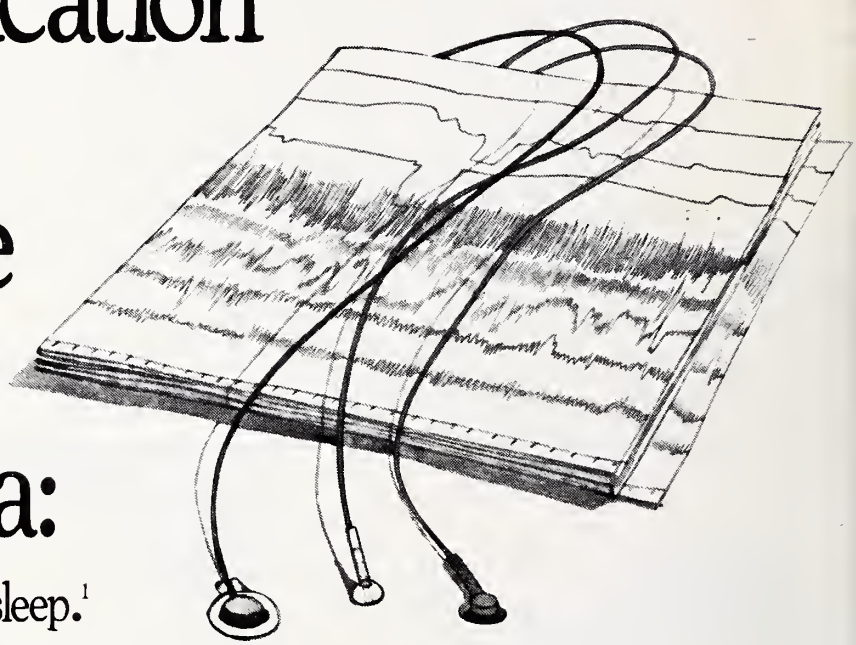
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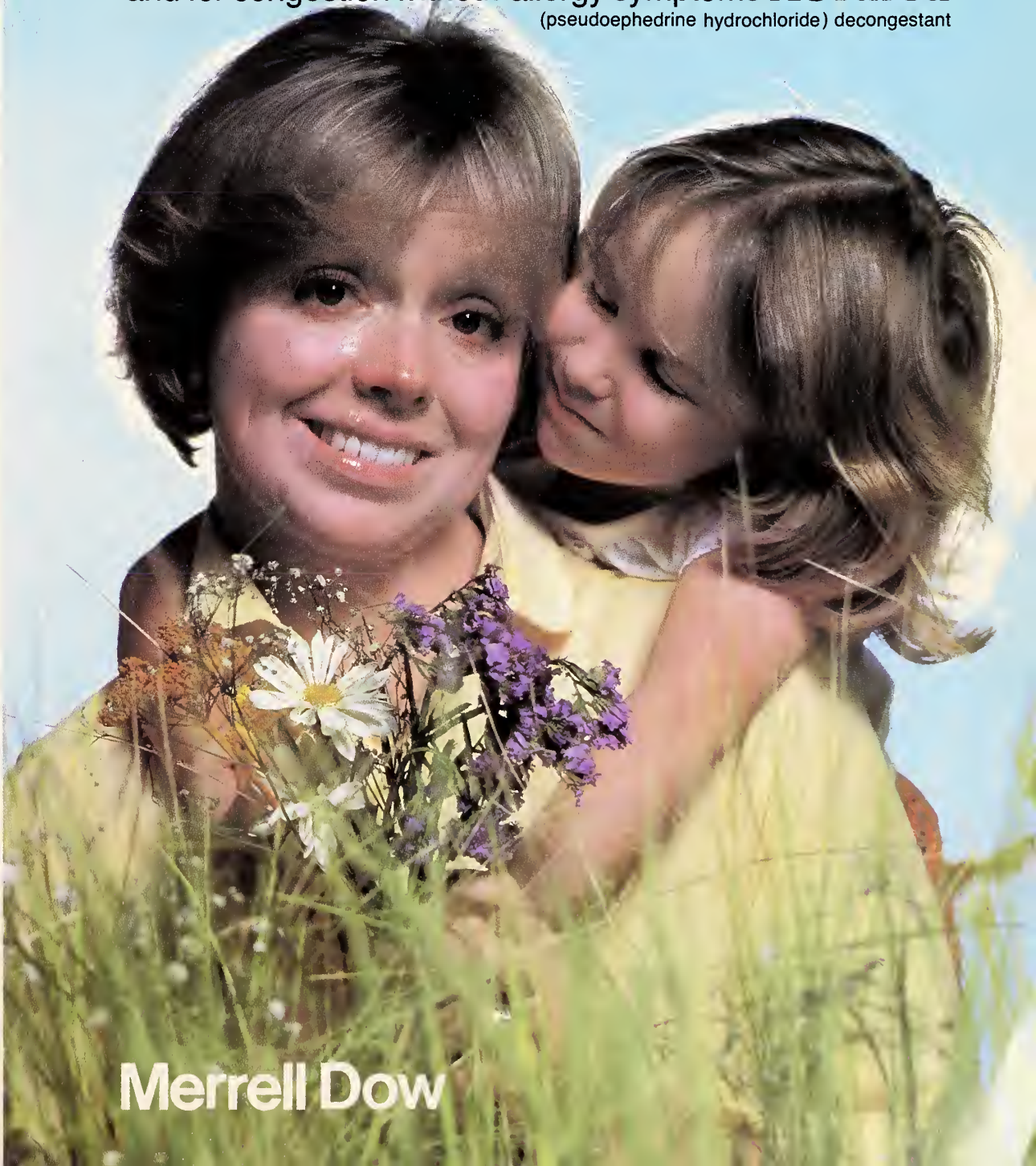
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The effectiveness of diazepam in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindications: Tablets or capsules in children under 6 months of age; known hypersensitivity; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because their use is rarely a matter of urgency and because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

ORAL: Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral forms adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures.

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Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status. Not recommended for OB use.

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects — particularly with known compounds which may potentiate action of diazepam, *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed and tolerated).

The clearance of diazepam and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

INJECTABLE: Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated.

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug.

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, observed in patients during and after diazepam therapy are of no known significance.

INJECTABLE: Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia.

In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported.

Dosage: Individualize for maximum beneficial effect.

ORAL: *Adults:* Anxiety disorders, relief of symptoms of anxiety—Valium (diazepam/Roche) tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 Valrelease capsules (15 to 30 mg) daily. Acute alcohol withdrawal—tablets, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; or 2 capsules (30 mg) the first 24 hours, then 1 capsule (15 mg) daily as needed. Adjunctively in skeletal muscle spasm—tablets, 2 to 10 mg t.i.d. or q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily. Adjunctively in convulsive disorders—tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily.

Geriatric or debilitated patients: Tablets—2 to 2½ mg 1 or 2 times daily initially, increasing as needed and tolerated (see Precautions). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose.

Children: Tablets—1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use in children under 6 months). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose (not for use in children under 6 months).

INJECTABLE: Usual initial dose in older children and adults is 2 to 20 mg I.M. or I.V., depending on indication and severity. Larger doses may be required in some conditions (tetanus). In acute conditions injection may be repeated within 1 hour, although interval of 3 to 4 hours is usually satisfactory. Lower doses (usually 2 to 5 mg) with slow dosage increase for elderly or debilitated patients and when sedative drugs are added. (See Warnings and Adverse Reactions.)

For dosages in infants and children see below; have resuscitative facilities available.

I.M. use: by deep injection into the muscle.

I.V. use: inject slowly, take at least one minute for each 5 mg (1 ml) given. Do not use small veins, i.e., *dorsum of hand or wrist. Use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.*

Moderate anxiety disorders and symptoms of anxiety, 2 to 5 mg I.M. or I.V., and severe anxiety disorders and symptoms of anxiety, 5 to 10 mg I.M. or I.V., repeat in 3 to 4 hours if necessary; acute alcohol withdrawal, 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary. Muscle spasm, in adults, 5 to 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary (tetanus may require larger doses); in children *administer I.V. slowly*; for tetanus in infants over 30 days of age, 1 to 2 mg I.M. or I.V., repeat every 3 to 4 hours if necessary; in children 5 years or older, 5 to 10 mg repeated every 3 to 4 hours as needed. Respiratory assistance should be available.

Status epilepticus, severe recurrent convulsive seizures (I.V. route preferred), 5 to 10 mg adult dose administered slowly, repeat at 10- to 15-minute intervals up to 30 mg maximum. Repeat in 2 to 4 hours if necessary, keeping in mind possibility of residual active metabolites. Use caution in presence of chronic lung disease or unstable cardiovascular status. Infants (over 30 days) and children (under 5 years), 0.2 to 0.5 mg slowly every 2 to 5 min., up to 5 mg (I.V. preferred). Children 5 years plus, 1 mg every 2 to 5 min., up to 10 mg (slow I.V. preferred); repeat in 2 to 4 hours if needed. EEG monitoring may be helpful.

In endoscopic procedures, titrate I.V. dosage to desired sedative response, generally 10 mg or less but up to 20 mg (if narcotics are omitted) immediately prior to procedure; if I.V. cannot be used, 5 to 10 mg I.M. approximately 30 minutes prior to procedure. As preoperative medication, 10 mg I.M.; in cardioversion, 5 to 15 mg I.V. within 5 to 10 minutes prior to procedure. Once acute symptomatology has been properly controlled with injectable form, patient may be placed on oral form if further treatment is required.

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension. Dialysis is of limited value.

How Supplied:

ORAL: Valium scored tablets — 2 mg, white; 5 mg, yellow; 10 mg, blue — bottles of 100 and 500; Prescription Paks of 50, available in trays of 10; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25 and in boxes containing 10 strips of 10.

Valrelease (diazepam/Roche) slow-release capsules — 15 mg (yellow and blue), bottles of 100; Prescription Paks of 30.

INJECTABLE: Ampuls, 2 ml, boxes of 10; Vials, 10 ml, boxes of 1; Tel-E-Ject® (disposable syringes), 2 ml, boxes of 10. Each ml contains 5 mg diazepam, compounded with 40% propylene glycol, 10% ethyl alcohol, 5% sodium benzoate and benzoic acid as buffers, and 1.5% benzyl alcohol as preservative.

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The abused child will grow up someday. Maybe.

Each year, over one million American children suffer from child abuse. And over 2,000 children die from it.

But what about those who survive?

Statistics show that an abused childhood can affect a person's entire life.

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The National Committee for Prevention of Child Abuse is a private, charitable organization that knows how to prevent child abuse.

But we need your help to do it.

We need your money. We need more volunteers.

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Because if we don't all start somewhere, we won't get anywhere.

National Committee for Prevention of Child Abuse

Help us get to the heart of the problem.

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Book REVIEWS

***PCP: The Devil's Dust — Recognition, Management, and Prevention of Phencyclidine Abuse,* by Ronald L. Linder with Steven E. Lerner and R. Stanley Burns. Wadsworth Publishing Company, Belmont, California, 1982. 176 pages.**

Phencyclidine — more commonly known as PCP, or "Angel Dust" — is, in terms of its effects, the most unpredictable of all street drugs. PCP is often misrepresented as mescaline, psilocybin, or LSD-25. The drug's effects can range from an exhilarating high to delirium. Since PCP causes different effects at varying dosages, it cannot accurately be placed in any drug category. Most quasi-experts call it a tranquilizer-anesthetic with psychedelic properties, although it is chemically unrelated to LSD-25. PCP is a very dangerous substance, with high potential for overdose.

Produced as a white, crystalline powder, phencyclidine can be combined with other drugs, or sprinkled on any smokable substance, such as tobacco, marijuana, or parsley. Parke-Davis first synthesized PCP in 1957. Although it was tested on humans for several years, the drug's side effects made it unsuitable for therapeutic use.

The effects that occur following ingestion depend on several variables. Although the drug's method of action is unclear, it appears to act on the sensory cortex, thalamus, and midbrain in such a manner as to "scramble" internal stimuli, thereby impairing perception. Effects on body and mind depend upon the user's psychopathology, dosage, method of ingestion, and circumstances under which PCP is taken, including setting, immediate feelings, and presence of others.

Ronald Linder, principal author of *PCP: The Devil's Dust,* is Assistant Director of the Department of Continuing Education in Health Sciences at the University of California, Los Angeles Extension. He also directs the California State PCP Abuse Training and Prevention Project. Steven Lerner is Project Coordinator for the same Project. A widely-published clinical researcher, Lerner has testified in many PCP-related murder cases. R. Stanley Burns is a Staff Neurologist with the National Institute of Mental Health who specializes in neurology and pharmacology. He has considerable clinical experience in drug abuse treatment and has published many PCP-related articles.

PCP: The Devil's Dust rests on several suppositions. The authors recognize that studying phencyclidine leads to a dilemma faced by all drug researchers; *i.e.*, historically in this country, the hazards of psychotropic drugs have been overstated and sensationalized. The best way to avoid this dilemma and to assess the true danger posed by a psychotropic substance is through systematic research into its effects on laboratory animals and human volunteers.

The authors contend that PCP differs markedly from other abused drugs. Despite absence of much research into its effects, the evidence of risk attending use that does exist is frightening. PCP represents a class of drugs with effects unlike those of other abused substances. Because the problems that phencyclidine poses are so new, the authors begin by summarizing in general terms how psychotropic drugs affect behavior. In order to recognize PCP abusers, one must understand what forms the drug can take on the street, its relative strengths and effects, as well as patterns of abuse. The authors provide guidelines for recognizing the PCP abuser, which are drawn from consultation with thousands of drug abuse experts.

PCP: The Devil's Dust begins by defining the problem. Included here is a discussion of how the problem developed, as well as an analysis of the drug's many identities. The book then discusses the extent of PCP abuse, its effects, and the PCP lifestyle. In Part Two the book explores how the PCP abuser can best be recognized and treated. Part Three, which deals with abuse prevention, discusses PCP and the law as it applies to manufacture, sale,

(Continued on page 233)

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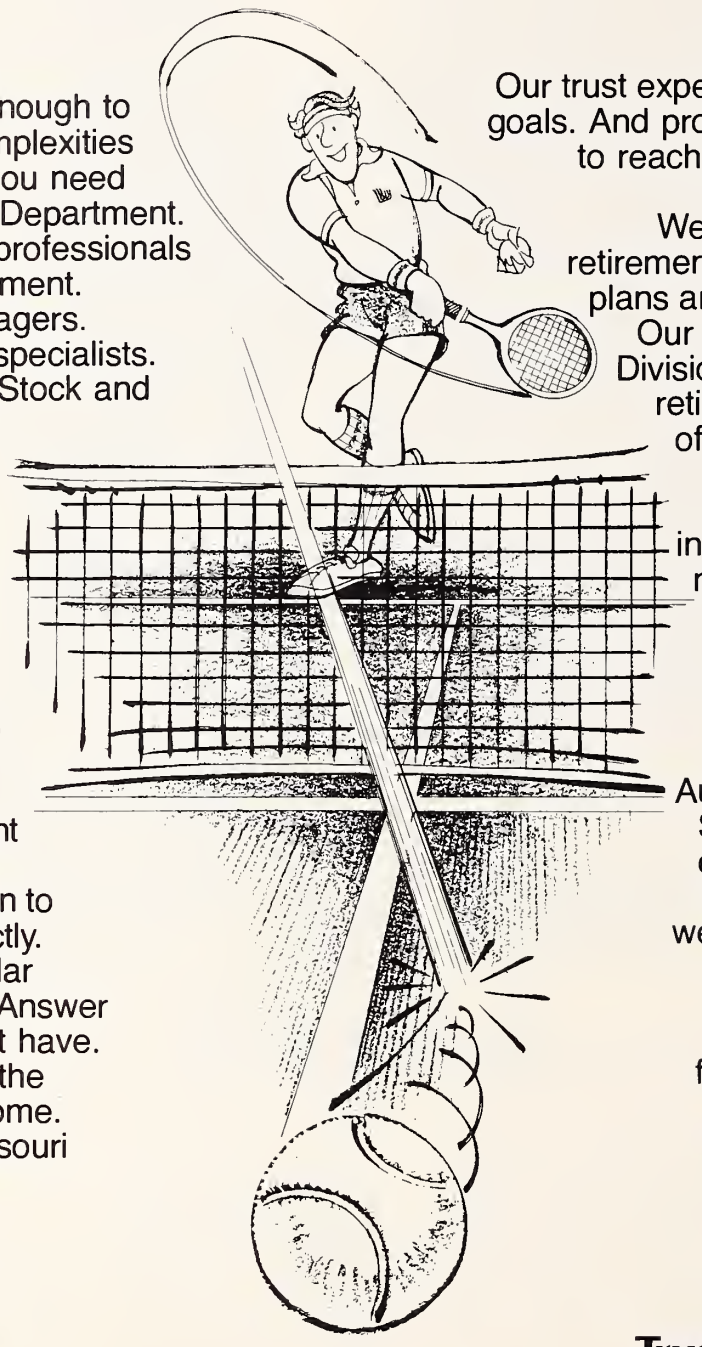
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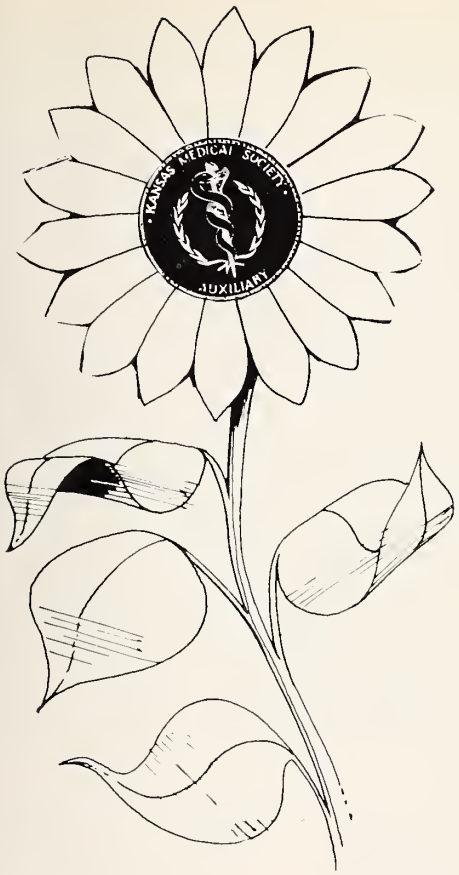
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AUXILIARY NEWS

An Open Letter to Kansas Physicians

Dear Doctor:

It is my privilege to serve as President of the Kansas Medical Society Auxiliary during the 1983-84 year. This page will continue to serve as one means for informing you of Auxiliary activities. I hope your *best* source of information will be your spouse who has been motivated by awareness of purpose and activities of the Auxiliary to become an active member of the county, state, and national organizations.

The response from Auxiliary members who were asked to chair committees and fill other key roles has been most gratifying. Everyone contacted was positive and enthusiastic about the goals and planned activities. With this support and group leadership, how can the Auxiliary do anything but grow in size and vitality?

A word to you Kansas "Lady Docs": This year the Auxiliary will launch a drive to recruit male spouses. There are 207 women physicians in Kansas, and last year (the most recent for which figures are available), we had only six male spouse members. Dr. Roger Warren, Hanover, a physician and also one of the mighty six male auxiliary members, has agreed to chair the Committee of Male Member Recruitment and Retention. We need the financial and personal support of these potential members, and we plan to widen our scope of activities to include the male spouses. Anthony J. Sanfelippo, Fond du Lac, Wisconsin, attorney, heads the national program. He has taken time from his practice to participate in Auxiliary activities at the National Convention and Fall Confluence.

Last year I accompanied Meldon Laury (David) on visits to the county auxiliaries. We had a glorious time and I learned a lot about the Kansas Auxiliary. Thanks, Meldon, for being a great teacher. Now, Ann Rempel (John) will join me in my wanderings. This is a great method to "spread The Word" about the programs of the Auxiliary, and for us, the officers, to meet a great group of Kansans.

One of the stated purposes of the KMSA is promotion of health education, and we will continue to work closely with both State and County Medical Societies and Auxiliaries in carrying out this purpose. Please alert me if there are services that we are neglecting. Also, let me know if there are related projects or organizations we could support or assist. This is the age of coalitions, and as was proven magnificently by the successful Salina conference, "An American Crisis: Wellness *vs* Abuse," a group of organizations can really make a project "fly." Credit for this is due to the superb organizational and coordinating skills of the Kansas Medical Society office and the guiding hands of Nancy Roderick (James) from the Auxiliary and Kermit Wedel, M.D., KMS President. Thanks and appreciation to all of these people.

Diane Sanders
President
Kansas Medical Society Auxiliary

GOOD NEWS!

You can **now** get our non-cancellable, guaranteed renewable disability plan at reduced premium rates and non-smoker rates.

This plan is endorsed by the Kansas Medical Society and especially designed to meet your needs.

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OFTEN INSEPARABLE: PAIN AND ANXIETY

A pathologic partnership one sees every day

Pain—triggering anxiety—which accentuates the perception of pain...together they're worse than either alone. And since they're usually both present in musculoskeletal disorders, the best therapy is often a combination of analgesic and anxiolytic agents. Equagesic ® -M combines the pain relief of aspirin with the tension-reducing properties of meprobamate—because together they're better than either alone.

See important information on next page.

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tablets

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Effective analgesic/anxiolytic alliance





Prompt, effective relief from the dual burden of pain and anxiety

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Effective analgesic/anxiolytic alliance

Proven superior to aspirin alone in controlled clinical trials

(BRIEF SUMMARY)

DESCRIPTION: Each tablet contains 200 mg meprobamate and 325 mg aspirin.

INDICATIONS: Adjunct in short-term treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease. Clinical trials demonstrated in these situations relief of pain is somewhat greater than with aspirin alone. Effectiveness in long-term use, i.e. over 4 months, has not been assessed by systematic clinical studies. Physicians should periodically reassess usefulness of drug for individual patients.

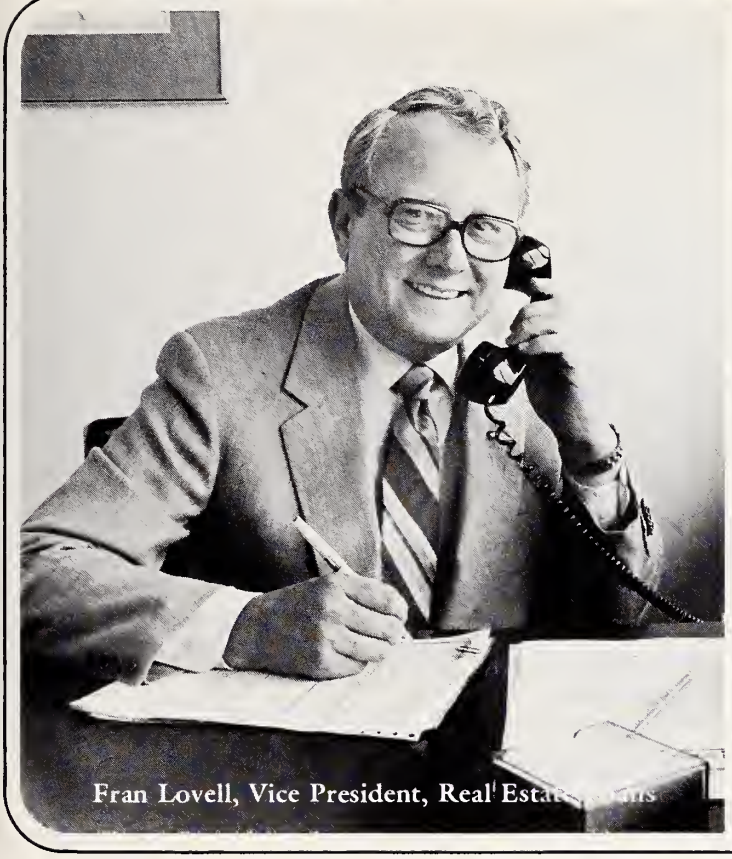
CONTRAINDICATIONS: ASPIRIN: Allergic or idiosyncratic reactions to aspirin or related compounds. MEPROBAMATE: Acute intermittent porphyria; allergic or idiosyncrotic reactions to meprobamate or related compounds, e.g. carisoprodol, mebutamate, or carbromal.

WARNINGS: ASPIRIN: Use salicylates with extreme caution in patients with peptic ulcer, asthma, coagulation abnormalities, hypoprothrombinemia, vitamin K deficiency, or those on anticoagulants. In rare instances, aspirin in persons allergic to salicylates may result in life-threatening allergic episodes.
MEPROBAMATE: DRUG DEPENDENCE: Physical and psychological dependence, and abuse have occurred.
Chronic intoxication from prolonged ingestion of, usually, greater than recommended doses is manifested by ataxia, slurred speech, and vertigo. Therefore, carefully supervise dose and amounts prescribed and avoid prolonged use, especially in alcoholics and others with known propensity for taking excessive quantities of drugs. Sudden withdrawal after prolonged and excessive use may precipitate recurrence of preexisting symptoms, e.g. anxiety, anorexia, or insomnia, or withdrawal reactions, e.g., vomiting, ataxia, tremors, muscle twitching, confusional states, hallucinosis, and, rarely, convulsive seizures. Such seizures are more likely in persons with CNS damage or preexistent or latent convulsive disorders. Onset of withdrawal symptoms occurs usually within 12 to 48 hours after discontinuation; symptoms usually cease within next 12-to-48-hour period. When excessive dosage has continued for weeks or months, reduce dosage gradually over 1 to 2 weeks rather than stop abruptly. Alternatively, a short-acting barbiturate may be substituted, then gradually withdrawn.
POTENTIALLY HAZARDOUS TASKS: Warn patients meprobamate may impair mental or physical abilities required for potentially hazardous tasks, e.g., driving or operating machinery.
ADDITIVE EFFECTS: Since CNS-suppressant effects of meprobamate and alcohol or meprobamate and other psychotropic drugs may be additive, exercise caution with patients taking more than one of these agents simultaneously.
USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during first trimester of pregnancy, has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at time of institution of therapy should be considered. Advise patients if they become pregnant during therapy or intend to become pregnant to communicate with their physicians about desirability of discontinuing the drug.
Meprobamate passes the placental barrier. It is present both in umbilical cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breastfeeding patients, consider the drug's higher concentrations in breast milk as compared to maternal plasma levels.
USAGE IN CHILDREN: Keep preparations with aspirin out of reach of children. Equagesic®-M is not recommended for patients 12 years of age and under.
PRECAUTIONS: ASPIRIN: Salicylates antagonize uricosuric activity of probenecid and sulfinpyrazone. Salicylates are reported to enhance hypoglycemic effect of sulfonylurea antidiabetics.
MEPROBAMATE: Use lowest effective dose, particularly in elderly and/or debilitated, to preclude over-sedation. Meprobamate is metabolized in the liver and excreted by the kidney; to avoid excess accumulation exercise caution in its use in patients with compromised liver or kidney function. Meprobamate occasionally may precipitate seizures in epileptic patients. It should be prescribed cautiously and in small quantities to patients with suicidal tendencies.
ADVERSE REACTIONS: ASPIRIN: May cause epigastric discomfort, nausea, and vomiting. Hypersensitivity reactions, including urticaria, angioneurotic edema, purpura, asthma, and anaphylaxis may rarely occur. Patients receiving large doses of salicylates may develop tinnitus.
MEPROBAMATE: CNS: Drowsiness, ataxia, dizziness, slurred speech, headache, vertigo, weakness, paresthesias, impairment of visual accommodation, euphoria, overstimulation, paradoxical excitement, fast EEG activity.
GI: Nausea, vomiting, diarrhea.
CARDIOVASCULAR: Palpitation, tachycardia, various forms of arrhythmia, transient ECG changes, syncope, hypotensive crisis.
ALLERGIC OR IDIOSYNCRATIC: Milder reactions are characterized by itchy, urticarial, or erythematous maculopapular rash, generalized or confined to the groin. Other reactions include leukopenia, acute nonthrombocytopenic purpura, petechiae, ecchymoses, eosinophilia, peripheral edema, adenopathy, fever, fixed drug eruption with cross-reaction to carisoprodol, and cross-sensitivity between meprobamate/mebutamate and meprobamate/carbromal. Rare, more severe hypersensitivity reactions include hyperpyrexia, chills, angioneurotic edema, bronchospasm, oliguria, and anuria. Also, anaphylaxis, exfoliative dermatitis, stomatitis, and proctitis. Stevens-Johnson syndrome and bullous dermatitis have occurred.
HEMATOLOGIC (SEE ALSO "ALLERGIC OR IDIOSYNCRATIC"): Agranulocytosis, aplastic anemia have been reported, although no causal relationship has been established, and thrombocytopenic purpura.
OTHER: Exacerbation of porphyric symptoms.
DOSAGE AND ADMINISTRATION: Usual dose is one or two tablets, 3 to 4 times daily as needed for relief of pain when tension or anxiety is present. Not recommended for patients 12 years of age and under.
OVERDOSAGE: Treatment is essentially symptomatic and supportive. Any drug remaining in the stomach should be removed. Induction of vomiting or gastric lavage may be indicated. Activated charcoal may reduce absorption of both aspirin and meprobamate. Aspirin overdosage produces usual symptoms and signs of salicylate intoxication. Observation and treatment should include management of hyperthermia, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions. Suicidal attempts with meprobamate have resulted in drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.
Some suicidal attempts have been fatal. The following data, reported in the literature and from other sources, are not expected to correlate with each case (considering factors such as individual susceptibility and length of time from ingestion to treatment), but represent usual ranges reported. Acute simple overdose (meprobamate alone): Death has been reported with ingestion of as little as 12 gram meprobamate and survival with as much as 40 gram.
BLOOD LEVELS:
0.5-2.0 mg percent represents usual blood-level range after therapeutic doses. The level may occasionally be as high as 3.0 mg percent.
3-10 mg percent usually corresponds to findings of mild-to-moderate symptoms of overdosage, such as stupor or light coma.
10-20 mg percent usually corresponds to deeper coma, requiring more intensive treatment. Some fatalities occur.
At levels greater than 20 mg percent, more fatalities than survivals can be expected.
Acute combined overdose (meprobamate with other psychotropic drugs or alcohol): Since effects can be additive, history of ingestion of a low dose of meprobamate plus any of these compounds (or of a relatively low blood or tissue level) cannot be used as a prognostic indicator.
In cases of excessive doses, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Any drug remaining in stomach should be removed and symptomatic treatment given. Should respiration or blood pressure become compromised, respiratory assistance, CNS stimulants, and pressor agents should be administered cautiously as indicated. Diuresis, osmotic (mannitol) diuresis, peritoneal dialysis, and hemodialysis have been used successfully in removing both aspirin and meprobamate. Alkalinization of the urine increases excretion of salicylates. Careful monitoring of urinary output is necessary, and caution should be taken to avoid overhydration.
Relapse and death, after initial recovery, have been attributed to incomplete gastric emptying and delayed absorption.
HOW SUPPLIED: Bottles of 50 scored tablets.

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Book Reviews

(Continued from page 229)

possession, and use. Included here is an analysis of what community actions can be taken in an effort to prevent PCP abuse, as well as how PCP intoxication can influence criminal proceedings.

Research for this book began in 1974, when Drs. Burns and Lerner were members of a federally-funded research team that was seeking to develop new treatment modalities for polydrug abusers. Their first contact with PCP habitues came while they were providing medical assistance during rock concerts. The book is based on a study of more than 2,000 PCP abusers. The authors purposely selected the most lurid stories in order to lend to the reading as much sensationalism as possible. This was done in the belief that the most dramatic examples best illustrate the areas of major PCP concern.

The authors conclude that the single greatest risk to the recreational PCP user is increasing that use until it becomes chronic. The drug remains in the body of the chronic PCPer for days, thereby increasing the chances of an unpredictable effect.

PCP experiences tend to evolve into a lifestyle with features unlike those surrounding other abused drugs. One distinctive feature of this lifestyle is the family-like atmosphere of the peer group. A sharing relationship seems to pervade the coterie. But, as heavy users increase their use of the drug, they become more isolated, in time divorcing themselves from the group as interpersonal relationships deteriorate.

The authors believe that no drug in so little time has taken a greater toll in life and suffering than has PCP. They further believe the most alarming aspect of phencyclidine abuse is the self-mutilation, suicide, and homocide that accompany it. PCP is costly to the community in terms of lost income, medical costs, crime resulting from the user's urgent need to support his/her habit, untaxed income resulting from the illegal manufacture of PCP, and harm to social workers who try to help.

PCP is a unique drug with unique effects and hazards. It presents an equally unique challenge for drug treatment personnel. Established methods of



helping substance abusers are ineffectual in dealing with PCPers. Most established programs for assisting drug abusers tend to focus on users of alcohol and opiates, or on polydrug abusers. PCP presents a different kind of problem. Drug treatment personnel need to recognize that traditional programs not only are ineffective in treating PCPers, but may actually aggravate the problem.

Criminal acts associated with PCP use have created problems for the criminal justice system. Few members of that system are familiar with the unique properties of PCP and its analogs and the major points that need to be established in litigating PCP-related criminal acts.

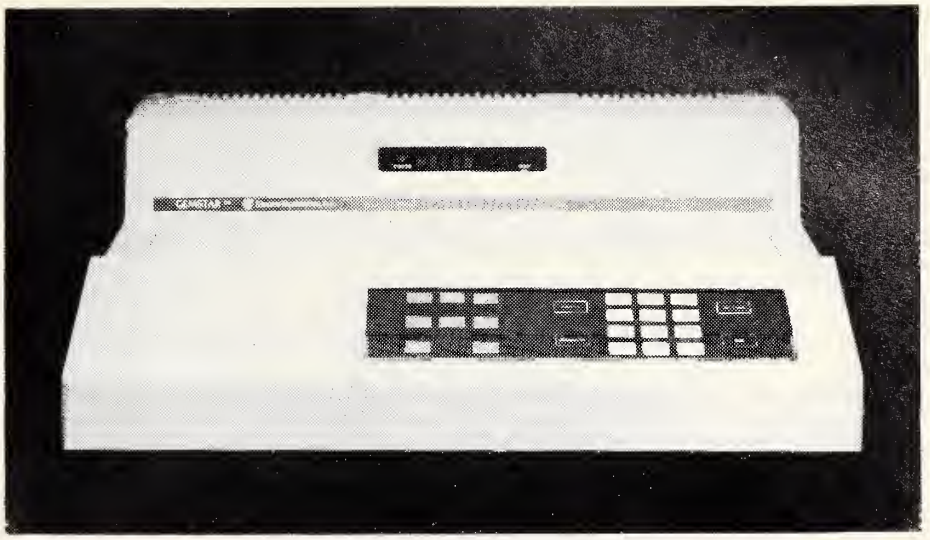
Linder *et al.* are convinced that most human problems, including drug abuse, cross traditional disciplinary boundaries. The solution to those problems, therefore, requires input from many disciplines. Drug abuse is both the cause and symptom of much human agony. The approach taken to it, however, has oftentimes been simplistic. Attempts to reduce substance abuse in a drug-oriented culture by outlawing drugs is futile. A realistic response to the drug problem involves looking at the whole person, with particular attention paid to forces that cause people to rely on drugs. A prerequisite to this collective response is educating substance abuse personnel to recognize that drug abuse can best be managed through a comprehensive, multicultural approach.

PCP abuse is not a single drug problem. Substance abuse is interwoven into an intricate fabric of human suffering, technology, and social institutions. PCP abuse is a dilemma that can best be stopped before it begins; *i.e.*, through prevention. Since reasons why people use drugs to alter consciousness are varied and complex, the means of prevention must be equally varied and complex. Some individuals will always use or abuse drugs. The authors of *PCP: The Devil's Dust* believe that if an individual is going to use drugs, s/he should do so knowledgeably, not ignorantly. This suggests that an intelligent response to the PCP problem will include dissemination of facts, both positive and negative, about use of the drug.

PCP: The Devil's Dust contains a useful glossary and bibliography, as well as a list of referral agencies

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in the states to which substance abusers can turn for help. The book wisely notes that the federal government's response to emerging PCP use — *i.e.*, making it a controlled drug — resulted in discontinuation, at a critical time, of ongoing clinical research into the pharmacology of PCP. As a consequence, basic pharmacological understanding was not available when the PCP epidemic struck, making recognition of acute PCP intoxication and effective treatment difficult, if not impossible.

PCP: The Devil's Dust is a valuable guide to help prepare professionals to treat PCP users and to reduce PCP hazards in high-risk settings.

The book has its weaknesses, and these include some undocumented comparative implications, simplistic explanations, and unwarranted conclusions.

For instance, it states that "In one study of chronic PCPers, only 57 per cent reported satisfactory sex lives." But what evidence is there that a larger segment of the American populace — those who do not use PCP — would report a satisfactory sex life? It is alleged that "The motive force behind PCP use seems to be the desire to escape." But a desire to escape can be used to explain (simplisticly) most types of negative human behavior. It is too broad an explanation to be meaningful.

In a discussion of PCP and the law, the authors observe that "The first group of drugs, called Schedule I in the 1970 Controlled Substances Act, includes such well-known hazardous substances as heroin and LSD." How hazardous is LSD-25? Placement of this psychedelic in Schedule I has prevented research from being conducted into its possible legitimate therapeutic uses. PCP is currently classified in Schedule II, which includes substances that have a high abuse potential and that produce psychological and/or physical dependence, but with an acceptable, albeit restricted, medical use. The authors believe PCP should be placed in Schedule I. "This change would raise the penalty for manufacture and sale from 5 to 15 years, adding a badly needed deterrent." It is debatable whether manufacturers and distributors of PCP would be any more deterred from manufacturing and distributing if the drug were placed in Schedule I. This criticism is supported by the authors' own observation that "trying to reduce drug abuse in a drug-oriented culture by outlawing drugs alone is like trying to

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prevent tooth decay by enacting a prohibition on candy.'' When the profit motive is alive and well, legal deterrents accomplish little, except to drive up costs of proscribed substances and thereby increase the profits that can be made from trading in contraband.

It is contended that ''Each drug problem has its own identity or culture — marijuana and jazz musicians, alcohol and skid row, cocaine and the entertainment industry. . . .'' This statement is preposterous. None of the drugs mentioned is today prominently identified with one specific culture. Marijuana, alcohol, and cocaine are all used by a broad cross-section of American society. The authors sum up their moralistic attitude regarding prevention of PCP abuse by arguing that youth today must take responsibility for humanity's future. ''If we are to survive ourselves, the 'me first' orientation of personal philosophy must yield to a 'we first' concept of life.'' Sure.

This book should be of interest to public health officials, psychologists, legislators, criminologists, and physicians. *PCP: The Devil's Dust* should also appeal to the concerned layperson. — *J.L.Z.*

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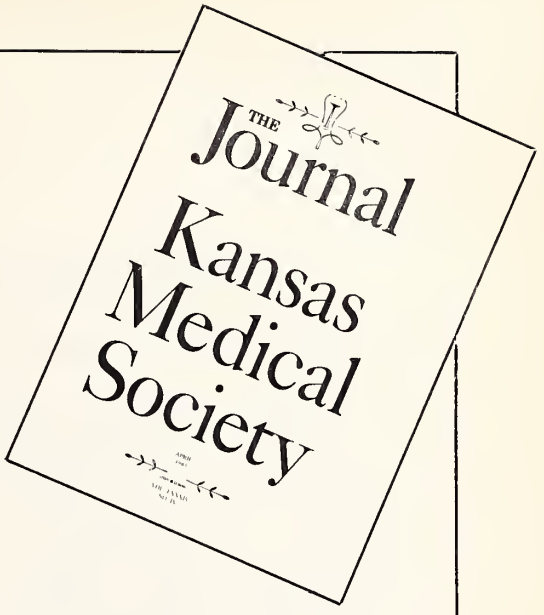
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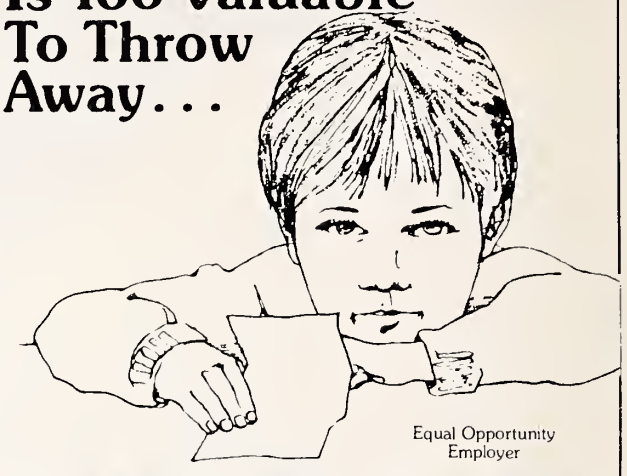
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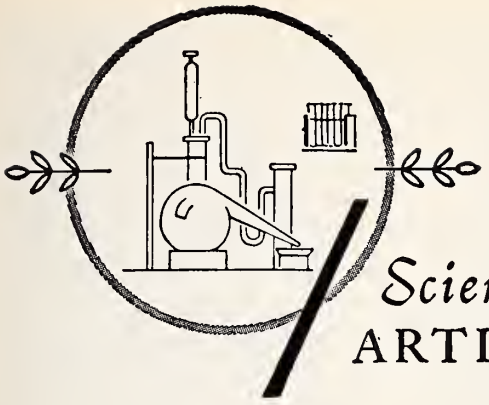
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Anorectal Abscesses

Extensive Manifestation with Abdominal Involvement

JOYCE MOORE STOVALL, M.D., *Leavenworth*

A DIABETIC male patient, 63 years old, was admitted for fever and abdominal pain associated with nausea, vomiting, and diarrhea of four days' duration. The patient stated that he had pain in his right buttock and right leg after a fall on stairs resulted in trauma to the right buttock two weeks prior to this hospital admission.

The patient was febrile on admission. A physical examination revealed the right lower quadrant of the abdomen to be tender to palpation, swollen, and erythematous. There was extensive right perirectal swelling, tenderness, and erythema. There was no fistula tract. Rectal examination showed the right side of the pelvis to be markedly tender and swollen. A complete blood count revealed elevation of white blood cells with a shift to the left. A radiograph of the abdomen revealed an ill-defined area of increased density within the region of the pelvic cavity with an accumulation of mottled gas shadows on the right side which extended for several centimeters below the right ischial tuberosity *(Figure 1)*. There was separation of the gas filled loops of bowel present throughout the abdomen suggestive of generalized peritonitis *(Figure 2)*. The patient's past medical history revealed that he had had an appendectomy when he was 17 years old.

On the fifth day of hospitalization he underwent an exploratory laparotomy at which time incision and drainage of a right perianal abscess, a right ischiorectal abscess, and an extensive right supralevator abscess were performed. The extensive supralevator abscess extended posteriorly to the retroperitoneal portion of the right lower quadrant of the abdomen. A diverting double barrel sigmoid colostomy was performed because of the extensive penetration of the combined anorectal abscesses which communicated with the retroperitoneal portion of the right lower quadrant of the abdomen. The patient tolerated the surgery well and was given intravenous antibiotics. A radiograph taken following surgery revealed a normal gas pattern *(Figure 3)*.

Address reprint requests to Dr. Stovall, Radiology Service, V.A. Medical Center, Leavenworth KS 66048.

A male diabetic patient presented with signs of systemic toxicity and pain in the right buttock and leg. A diverting double barrel sigmoid colostomy was performed because of extensive penetration of a right perianal abscess, right ischiorectal abscess, and a right supralevator abscess which communicated with the retroperitoneal portion of the right lower quadrant of the abdomen. Five types of bacteria were cultured from these anorectal abscesses.

Discussion

One of the most serious manifestations of anal infection is anorectal abscess. Although the etiology of the anorectal abscess remains uncertain, it has been frequently theorized that most, if not all, anorectal abscesses originate in the anal glands located in the intersphincter space *(Figure 4)*. Inadequate drainage of the anal gland, caused either by



Figure 1. There is a long radiolucent shadow along the right side of the pelvic cavity which extends below the right ischial tuberosity. This shadow may be confused with that of a fluid filled strangulated loop of bowel or a tumor.

a decrease in the activity of the anal glands or poor drainage through their ducts, promotes the spread of infection through anal tissue. When the infection penetrates through the intersphincteric space to the adjacent perianal space or ischiorectal fossa, typical perianal abscess or ischiorectal abscess is formed *(Figures 4, 5)*. Anatomically, the perianal abscess is the most common with ischiorectal abscess being second. Other anatomical types of anorectal abscesses are submucous and supralevator *(Figure 5)*. Submucous or intramural abscesses result from the spread of infection along the rectal wall beneath the mucosa. The supralevator or pelvirectal abscess is the least common, and results when infected material burrows through the sphincter into the space superior to the levator ani muscle *(Figure 4)*.

Anorectal abscess may occur unilaterally or bilaterally. A bilateral ischiorectal abscess is called a "horseshoe" abscess. There may be a combination of anorectal abscesses as in this case. The blackened areas in *Figure 5* illustrate this.

Anorectal abscesses seem to have more predilection for males than females with a 2.5:1.0 ratio. Abscess occurs frequently in healthy adult males in their fourth and fifth decades of life. Diabetes mellitus seems to be the most common medical problem concurring with abscess.

If signs of systemic toxicity and pain are present but the abscess cannot be visualized or palpated, a deep or high anorectal abscess such as ischiorectal abscess or the less common supralevator abscess must be considered. Deep or high anorectal abscesses can become extensive. They can extend upward to the superior aspect of the supralevator space and communicate with the lower abdominal cavity as well as downward to the perineal skin. Because the ischiorectal abscess is deep and the supralevator

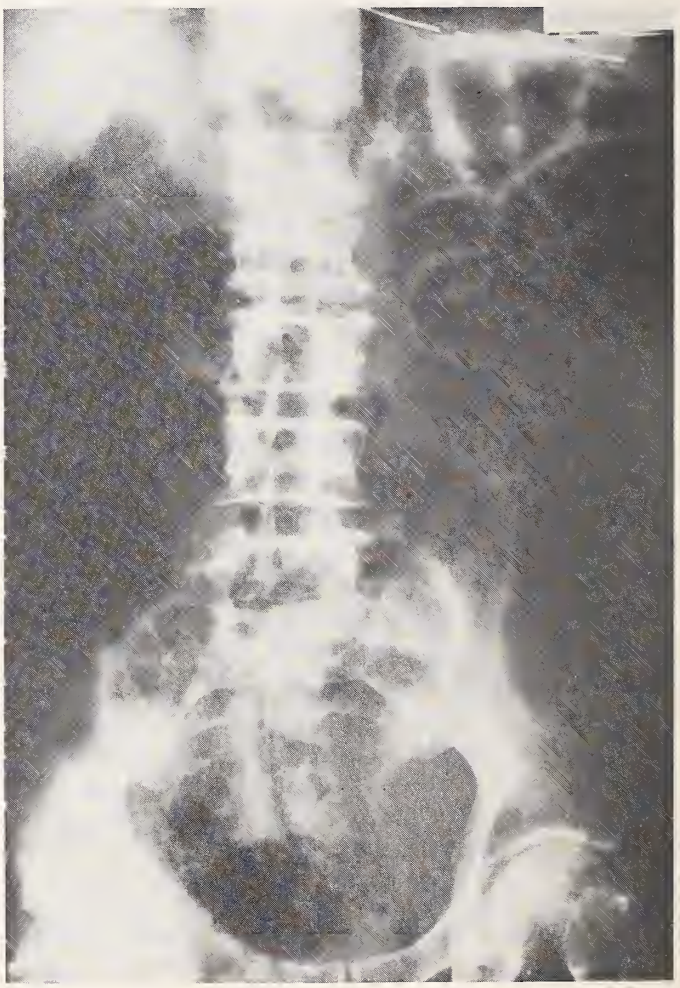


Figure 2. There is separation of gas-filled loops of bowel which is a sign of generalized peritonitis. Again present is the large accumulation of mottled gas shadows seen in the region of the pelvic cavity on the right side.



Figure 3. This radiograph was taken following surgery. There is no evidence of generalized peritonitis or the abscess shadow.



Figure 5. The lettered areas illustrate the different anatomical sites in which anorectal abscesses can occur. A. Ischiorectal abscess. B. Perianal abscess. C. Submucous or intramural abscess. D. Supralevator or pelvirectal abscess. The black areas represent the combination of anorectal abscesses involved in this case. All anatomical types are involved except for C.

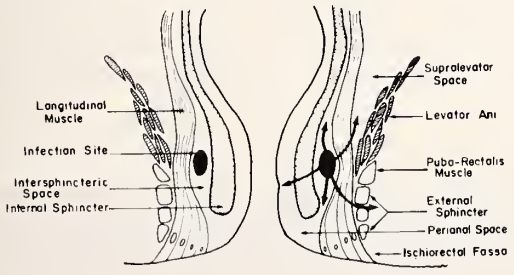


Figure 4. The left side of the illustration shows the origin of infection in the intersphincteric space which is located between the internal sphincter and the longitudinal intersphincteric muscle fibers. The right side illustrates various pathways in which an anorectal infection can spread from a primary focus in the intersphincteric space.

abscess is high, induration and erythema are observed later than with superficial perianal abscess. Patients with extensive anorectal abscesses should be hospitalized for appropriate treatment.

Several disorders are to be considered in the differential diagnosis of anorectal abscesses: pilonidal abscess, folliculitis, suppurative hidradenitis, periprostatic abscess, Bartholin gland abscess, and infected sebaceous cyst of the perianal skin. Rarely, conditions such as actinomycosis or tuberculosis require differentiation.

The patient presented in this case was diabetic and this probably predisposed him to this infection. Bacteria cultured were Citrobacter freundii, Escherichia coli, Klebsiella pneumoniae, Enterococcus, and Corynebacterium species.

Penile Implants

Surgical Treatment of Impotence

JOHN W. WEIGEL, M.D., *Kansas City, Kansas*

SEXUAL FUNCTION disorders in the male are no longer "closet diseases," and physicians must be prepared to discuss these problems with the patient, and in many cases his wife or sexual partner, and to initiate evaluation and arrange for appropriate therapy. It is now quite evident that most impotence problems are due to organic factors rather than emotional or psychogenic causes. Frequently both are present. With aging, the incidence of vascular disease, diabetes, and neurologic disorders rapidly increases as does use of medications — particularly those for control of hypertension which may affect both erectile and ejaculatory ability. Decreases in testosterone level are not well understood and may play a part. The incidence of prolactin secreting tumors seems to increase to a certain extent with age. Elevated levels of prolactin are known to interfere with erectile ability. These patients usually have a significantly depressed testosterone level. Many patients have multiple factors contributing to impotence and may have both psychogenic and organic causes. Patients have come to expect reasonably normal sexual function as part of their overall health in today's society, and they resent avoidance or superficial attitudes on the part of the physician toward these problems.

Normal sexual function in the male is composed of five integrated phenomena — erection, emission, ejaculation, orgasm, and detumescence. Erectile and ejaculatory impotence may co-exist, or may present as isolated entities. It is extremely important in obtaining the history to discuss these functions in some detail. Erectile response requires intact blood supply, innervation, and erectile tissue.

The blood supply to the penis is via the internal pudendal arteries — branches of the hypogastric arteries. Both large and small vessel disease may reduce blood flow.

Innervation essential to erection is provided by parasympathetics S2-4 via the pelvic nerves. Sympathetics — usually T9, 10-L2 — provide for emission through their effect on the muscles involving the epididymis, vas deferens, seminal vesicles, and prostate. Penile sensation and many of the muscles providing for propulsion of the spermatic fluid are provided by the pudendal nerve, a somatic nerve originating at S2-4.

The erectile tissue must be patent and compliant allowing tumescence to occur, and there must be adequate laxity of the tunics to expand significantly.

Impotence is a common and serious disorder affecting an increasing number of men as life expectancy is extended. It is common in individuals with diabetes and obstructive vascular disease, and is a frequent side effect of medications used to treat hypertension. Medical and, in particular, surgical advances have made treatment of impotence successful in most cases.

Erectile problems may consist of loss of erectile ability, diminution in quality or rigidity (partial erection), or the inability to maintain what may initially be an adequate erection.

Ejaculatory dysfunction includes retrograde ejaculation — extremely common in post prostatectomy patients, and frequently occurring in diabetics; and lack of ejaculation as in a patient who has had damage to the abdominal sympathetic nerve supply.

Important features in history taking include: quality, duration of erection; presence or absence of nocturnal erections; ability to ejaculate with or without a full erection; sexual dysfunction during masturbation or with different sexual partners; recent or remote onset, possible psychogenic factors; current or past illnesses, operations, medications, smoking, and drinking habits; and family history of diabetes. This information can usually be obtained in a discrete and unobtrusive manner by the examining physician as these patients are anxious to provide any facts that may be of help.

Physical examination should emphasize: body habitus — evidence of feminization in contour, breast development, hair distribution, fat, and muscle distribution; genital development; pulses in the lower extremities; sensory or reflex changes in the

Address reprint requests to Dr. Weigel, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.

lower extremities; and rectal examination, emphasizing sphincteric tone (S2-4), possible rectal or prostatic disease.

Laboratory evaluation should include the following studies: fasting blood sugar, with glucose tolerance test if indicated; BUN, creatinine; testosterone; and prolactin.

If the etiology is in doubt, adjunctive examinations may include Doppler examination of penile blood flow; penile blood pressure; bulbocavernosa reflex latency time; nocturnal penile tumescence studies; and psychological tests.

This approach is designed to detect the more common causes of both erectile and ejaculatory impotence. The more common entities include: vascular and neurologic conditions, psychogenic causes, endocrine disorders, diabetes, renal failure, and side effects of drugs and medications.

Treatment

Psychological disorders obviously require skilled therapy directed by qualified personnel. The past few years have seen the rapid proliferation of sexual therapists with various backgrounds. Competence and experience in this area are obvious prerequisites, and such individuals can provide an excellent service to these patients.

Effective medical therapy may be difficult to achieve, and in fact, the treatment of such diseases as hypertension is responsible for a great deal of sexual dysfunction arising from side effects of medications. Spironolactone is a prime example, and most of the antihypertensive medications have been felt to occasionally include these side effects. Changing drug programs may help.

Testosterone has frequently been employed on a trial basis. The patient's vascular status should be evaluated, and careful prostatic examinations done at six month intervals.

Prolactin secreting micro or macro-adenomas are an extremely uncommon finding in the author's experience. When present, surgery or bromocriptine therapy is required.

Surgical Therapy

Surgical approaches to erectile impotence are not new. Based on the fact that in many mammalian species the male has an os penis or baculum supplying rigidity, Bogoras used a segment of rib in 1936 to treat erectile impotence.[1] Bergman employed this technique in 1948 with limited success.[2] Goodwin and Scott used acrylic prosthetic material in 1952.[3] These devices were plagued with problems of erosion, pain, migration of the devices, and problems secondary to materials used. The development of medical grade silicone allowed penile implant surgery to develop into a safe and useful procedure. Lash introduced a solid silicone implant in 1964,[4] and this was followed by a modification designed by Pearman the following year.[5] These devices were bulky, were placed in the midline, and were designed to hinge against the pubis during intercourse. Pain, erosion, and device migration remained as problems. Morales conceived the use of paired rods in the corpora cavernosa in 1973,[6] and this concept was developed and popularized by Small, Carrion, and Gordon in 1975[7] *(Figure 1)*. Finney felt that the solid silicone prostheses had the potential for being too flexible for intercourse if they were to be cosmetically acceptable, and he constructed paired rods with a soft segment at the base of the penis to allow a

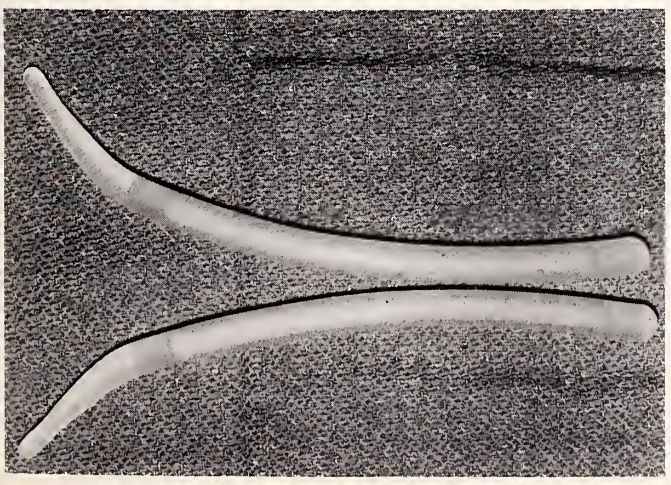


Figure 1. Small Carrion silicone penile implant.



Figure 2. Finney silicone penile implant with hinge effect.

hinge effect[8] *(Figure 2)*. This permitted firmness distal to the hinge.

Jonas employed the use of braided silver wire within paired silicone rods to permit rigidity, and to allow the prosthesis to remain in various positions enhancing cosmetic appearance[9] *(Figure 3)*. He was unable to show metallic fatigue, fraying, or breakage of the metallic strands.

In 1973, Scott and others reported on the use of an inflatable penile implant made of silicone consisting of a hollow fluid reservoir, a pump device, and hollow cylinders which were inserted into the corpora cavernosa.[10] Modifications of the various components have taken place since that time, and this device enjoys widespread usage at this time *(Figure 4)*. Advantages include the ability to achieve erection upon demand with avoidance of the possible cosmetic problems often associated with the semi-rigid rod type prosthesis. Better quality erections are possible. Disadvantages include higher costs, more difficult surgery, and a mechanical malfunction rate variously estimated at 5-40 per cent.

Patient acceptance in general has been excellent with the various types of prostheses. Most patients have significant pain during the early postoperative period, and residual soreness for several weeks is a common finding. There is a risk of infection of probably 3-5 per cent, highest in diabetics. If this occurs, the device usually must be removed. Repeat reimplantation can be done three months later in some cases.

Acceptance by sexual partners depends to a significant extent on preoperative counseling with both partners, and a thorough explanation of the surgery involved, the risks, and the functional aspects of the devices.

Thorough urologic evaluation is mandatory, particularly in patients with neurologic disease with voiding disorders. Implantation does not preclude self catheterization or urethral instrumentation.

The author has inserted penile implants in approximately 200 patients. A few patients with semi-rigid implants have complained of inadequate rigidity. Replacement with an inflatable implant has proven successful in that group. Device failure was much more common initially in the patients with inflatable penile implants. Improvements in the device and acquisition of surgical experience have reduced this problem significantly. Cylinder leakage is the most common problem followed by aneurysm formation at the base of the cylinder. These can be minimized with careful surgical technique and with patient compliance in avoiding over-inflation of the cylinders.

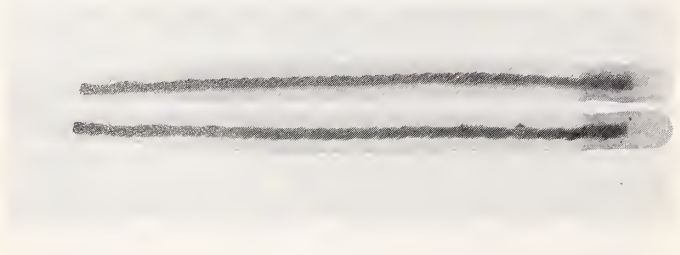


Figure 3. Jonas silicone penile implant with braided silver wire core.

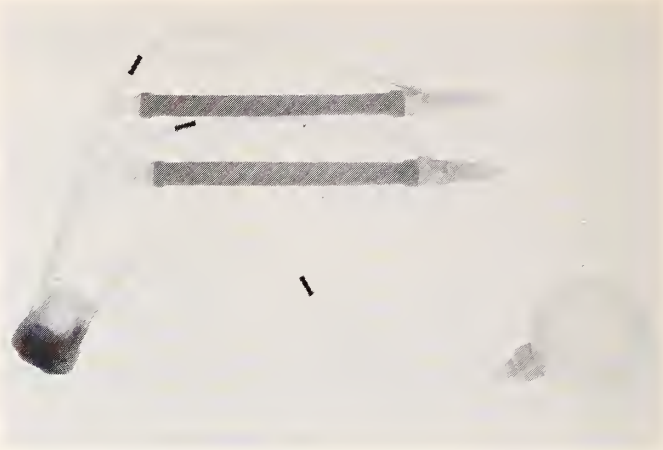


Figure 4. Scott inflatable penile implant — cylinders, reservoir, and pump.

Ejaculatory impotence may be successfully treated medically in some cases by stimulation of the alpha adrenergic receptors in the bladder neck and posterior urethra with ephedrine or with similar drugs. This is particularly helpful in diabetes. Patients with loss of ejaculation following damage to the sympathetics after lymphadenectomy rarely respond to this or to surgical modalities. Operative procedures designed to narrow the bladder neck have been successful in a few cases of retrograde ejaculation, particularly in patients who have had bladder neck procedures during childhood.

Conclusions

Surgical techniques are quite successful in the treatment of erectile impotence in many patients who have not responded to conservative therapy. There are advantages and disadvantages in both the semi-rigid and the inflatable penile implants. Bladder neck surgery is useful in the management of certain patients with bladder neck incompetence and ejaculatory impotence.

(Continued on page 284)

Radiation Therapy

Its Role in the Treatment of Early Breast Cancer

CARL M. MANSFIELD, M.D. *and* EASHWER K. REDDY, M.D., *Kansas City, Kansas*

THE HISTORY of breast cancer extends back for more than 5000 years. During this time, treatment became progressively more radical as it evolved from burning out the cancer with a hot stick to the supraradical mastectomy.[1] As each method failed, a more radical procedure replaced it. There is now a trend to less radical procedures in that there has been a definite move from the radical mastectomy toward the modified radical mastectomy. In recent years there has been substantial controversy regarding the primary treatment for early breast cancer. Discussion of this topic is especially important in the face of media coverage and legislative interest in informed consent laws. This article is written to present a position that deals with the role of radiation therapy in the treatment of early lesions that are 5 cm or less in diameter (T1-T2).

Technique

When breast cancer is treated by radiotherapy for cure, an attempt is made to treat tumor volume, adjacent normal tissue, and regional nodes with the least amount of damage to normal tissue. Although this approach is similar to that of curative surgery, it is possible to treat a larger volume of potential disease by radiotherapy than is routinely treated by mastectomy in that the internal mammary and supraclavicular nodes can be included.

Various radiation techniques have been used to deliver a tumoricidal dose to the treatment volume.[2] The breast is treated by tangentially opposed fields to include the breast and chest wall. Internal mammary and supraclavicular areas are treated by separate or combined direct anterior fields. The lower portion of the axilla is included in the tangential breast fields. The upper axilla is included in the supraclavicular field or in an additional posterior field. The entire treatment volume is given 4500-5000 rads in 5.5-6.0 weeks. The tumor "bed" is given an additional 1500-2000 rads by means of a small field of external irradiation or temporary implants of radioactive material.

Address reprint requests to Dr. Mansfield, Department of Radiation Therapy, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.

Results

Since 1976, at the University of Kansas, we have been treating a selected group of patients using lumpectomy and radiation therapy. The technique has been similar to that described here.

Patients have tolerated this procedure without complications. Cosmetic results have been excellent

Patients with stage I or II carcinoma of the breast can be treated by conservative surgery (lumpectomy) and radiation therapy. These patients can expect five and ten year disease free survival rates equal to those who undergo radical or modified radical mastectomies.

(Figure 1). Because of the success of this approach, there has been an increase in the number of patients referred for this procedure.

Discussion

Since 1927 there have been 25 major studies published of 5,314 patients who received curative treatment by lumpectomy, radiation therapy, or both as the only method of treatment. All but two reported survival statistics that were similar to those treated by various forms of mastectomy.[3]

In 1929 there were published results of the use of conservative surgery (tumorectomy) and radiation showing a five year survival rate of 60-70 per cent for early cases.[4, 5] In 1932, Pfahler reported a ten year survival rate of 83 per cent in 53 patients treated by radiotherapy.[6]

Porritt reported on 263 patients with early breast cancer randomized between radical mastectomy with occasional radiation therapy and local excision plus radiation therapy. Survival data favored the latter group.[7] Local excision plus radiation therapy patients had an 85 per cent (26/30) five year and a 70 per cent (9/13) ten year survival. Radical mastectomy patients had a five year survival rate of 60 per cent (30/50) and a ten year survival rate of 38 per cent (7/18). Peters[8] compared the results of 200 patients treated by excisional biopsy and radical



Figure 1. This patient is free of disease 15 years after lumpectomy and radiation therapy.

radiation therapy with 652 treated by radical mastectomy and radiation therapy. There was no difference in survival rates between the two groups. The five and ten year survival rates for both groups were 74 and 48 per cent respectively. Rissanen[9] retrospectively compared 1,008 cases of Stage I disease, 415 of whom were treated by local excision plus radiation therapy and 593 treated by radical mastectomy and radiation therapy. The five and ten year survival rates for the former group were 79 and 71 per cent respectively, and for the latter group, 82 and 71.5 per cent. The local excision group had a higher recurrence rate (25.8%); these patients were treated with additional radiation therapy or surgery. It is important to note here that although the recurrence rate was high, surgical salvage resulted in an excellent survival rate.

Mustakallio[10] was probably the second best known therapist who has advocated local excision or segmental resection plus radiation therapy. He reported Stage I results of 79 per cent (553/702) at five years, 61 per cent (257/418) at ten years, and 47 per cent (107/227) at 15 years.

Schlienger and Calle[11] reviewed 75 patients with tumors measuring less than 3 cm and no positive nodes treated by lumpectomy and radiation therapy. They had a five year disease free survival rate of 70.6 per cent (53/75); additional surgical salvage of eight recurrent cases raised the five year disease free survival to 80 per cent (61/75). Atkins[12] reported a ten year randomized trial comparing lumpectomy and radiation *vs* radical mastectomy and radiation therapy. Results in Stage I disease were comparable at five and ten years (80% for lumpectomy and 77% for radical at five years; 69% lumpectomy and 73% for radical at ten years). This randomized series clearly shows the effectiveness of this technique for Stage I disease. Results in Stage II disease were worse for lumpectomy at ten years (61% for lumpectomy and 73% for radical at five years; 23% for lumpectomy and 67% for radical at ten years). The low dose of radiation (3800 rads) given to the Stage II cases has been suggested as an explanation for results at ten years that are contrary to other reported results.[9-14]

For example, Papillon[15] noted a five year survival rate of 70 per cent for 191 early cases treated by lumpectomy and radiation therapy, and a ten year survival rate of 62 per cent. Also, in 1976 Cope *et al.*[16] reviewed a small group of patients treated by lumpectomy and radiation and noted a five year absolute survival of 83 per cent for Stage I and 73 per cent for Stage II, and no evidence of disease (NED) survival rate of 66 and 43 per cent respectively. Prosnitz *et al.*[17, 18] in a retrospective review of 150 patients from several centers, noted a NED five and ten year survival rate of 91 and 71 per cent respectively for Stage I and 60 and 40 per cent respectively for Stage II disease. Finally, Amalric *et al.*[19] noted a 77 per cent NED survival rate for T1-T2 N0 and 63 per cent for T1-T2 N2 at ten years in 1,424 cases.

The recurrence rate in breast cancer has been reported to be between 10 and 25 per cent, with the average being 10 per cent.[8, 9, 11] Recurrence can be managed by repeat local excision, by local irradiation, or by both. The preferred method is simple mastectomy. Using simple mastectomy, it has been possible to salvage 60-80 per cent of patients with local recurrences.

Summary

There is growing evidence in the literature that radiation therapy plus lumpectomy produce cure rates for Stage I and II disease equal to those of surgery.

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Bezoars

A Complication of Diabetic Gastroparesis

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ALTERATIONS in gastric motility and function are well documented in individuals with complicated diabetes mellitus. However, the prevalence of this condition is highly variable, with estimates ranging from 14 to almost 30 per cent.[1-3] No large prospective studies are available. Further, diagnosis is often difficult because of the need to employ invasive diagnostic techniques. It has been appreciated only recently that there is a clear association between diabetic gastroparesis and phytobezoars, but reports have been infrequent.[4, 5] We present here detailed information on five insulin-dependent individuals with one or more bezoars seen during the past three years at the University of Kansas Medical Center.

Clinical Data

Pertinent data concerning these five patients are listed in *Table I*. Three were female and two were male. All had insulin-dependent diabetes with peripheral neuropathy. None had had prior gastric surgery. Upper gastrointestinal series yielded normal results in three patients, were not done in one, and showed only thickened duodenal folds in the fifth patient. Bezoars were not detected in any patient radiographically. Upper gastrointestinal endoscopy was performed for various reasons and this was the means by which the bezoars were found. Mechanical disruption of the bezoars was then accomplished in all cases under endoscopic visualization.

Comment

Bezoars are persistent aggregates of particulate material found generally in the stomach. They may be composed primarily of vegetable fiber (phytobezoar), hair (trichobezoar) or even pills. A number of factors may predispose to their formation *(Table II)*. Common features in all these situations are a bulky nidus of material and delayed gastric emptying, the latter due either to local or systemic problems. Resultant symptoms range from minimal to severe and include pain, nausea and vomiting, bloating, early satiety or loss of appetite and, on occasion, gastrointestinal bleeding.

Delayed gastric emptying is a well-recognized feature of diabetes mellitus. It may be accompanied by the formation of phytobezoars as illustrated by this report of five patients followed during the past five years. Disturbances of both gastrointestinal function and glycemic control attributed to autonomic neuropathy may be due to bezoars, which can be reliably diagnosed only with endoscopy.

Involvement of the gastrointestinal tract as a manifestation of diabetic neuropathy has been recognized for years with stomach dysfunction being first described in 1945.[6] A more detailed inquiry into the problem and the first use of the term "gastroparesis diabeticorum" appeared in 1958.[2] As indicated earlier, accurate estimates of the incidence of the condition are unavailable due to the lack of specificity of the symptoms and to methodological difficulty in diagnosis. Barium studies have been shown to be inadequate indicators of the functional state of the stomach.[7] Currently employed techniques involve measurement of the disappearance of a radioisotopically labeled meal. Unfortunately, the substances administered are mostly liquid, and while movement of liquids seems to assess adequately the physiologic state of the gastric fundus, their use does not reflect antral activity which requires solids. The technique used in our patients involved a mixed test meal, including powdered eggs, whole milk, and instant breakfast, containing 50 $\mu Cu/^{113m}$ indium chloride.[8] Since this test meal consists of both solids and liquids, it is felt to be more representative of gastric function under normal feeding conditions. However, it is important to note that even with this more sophisticated test meal, two of our patients with bezoars had gastric emptying time within the normal range.

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TABLE I
CLINICAL DATA

Age	*Sex*	*Diabetes Duration (yrs)*	*Chief GI Symptoms*	*Dysphagia*	*Delayed Gastric Emptying*	*Diarrhea*	*Bladder Dysfunction*	*Postural Hypertension*	*Impotence*
23	M	5	Diarrhea	−	+	+	−	−	−
43	F	9	None	−	−	−	−	−	N/A
37	F	30	Nausea	−	−	+	−	+	N/A
63	M	20	Heartburn	+	+	−	−	+	+
32	F	20	Nausea	−	+	+	−	+	N/A

+ present − absent N/A not applicable.

A major consequence of diabetic gastroparesis is difficulty in achieving glycemic control. Erratic delivery of gastric contents into the small bowel can result in wide swings in plasma glucose levels, especially in patients on single dose intermediate-acting insulin. Nerve conduction velocity is partially dependent on tightness of glucose control, and normalization of plasma glucose can actually improve nerve conduction velocity.[9] Thus a vicious cycle can ensue with worsened control leading to aggravated neuropathy which further exacerbates the gastroparesis. It is obvious why symptomatic gastroparesis has such a dismal prognosis in that one-third of affected individuals die within three years.[10] However, it is conceivable that many symptoms attributed to gastric neuropathy are actually due to bezoars. A proper diagnosis is critical since bezoar disruption may give considerable symptomatic relief as it did in two of our patients with gastrointestinal complaints.

It is also important to recognize that virtually all patients with autonomic neuropathy have peripheral neuropathy as well *(Table I)*. Many drugs used to relieve symptoms of painful neuropathy — such as codeine, benzodiazepines, and tricyclic antidepressants — have an adverse effect on gastric motility and can exacerbate diabetic gastroparesis and enhance bezoar formation. The current popularity of high fiber diets as an adjunct to diabetic treatment might also increase the frequency of bezoars.[11]

Therapy of gastroparesis is difficult. Removal of bezoars may provide relief of upper gastrointestinal symptoms, but there is no overall effect demonstrable on gastric motility. Treatment with metoclopramide, a dopamine antagonist, has shown promise. The drug is known to increase the number and strength of gastric antral contractions, and should theoretically facilitate solid food disposal.[12] Despite its use, however, two of our patients have had recurrent bezoars.

We believe the diagnosis of bezoar should be considered in a diabetic patient who presents with any of the above mentioned gastrointestinal complaints. It should also be considered in any insulin-dependent diabetic in whom glycemic control is difficult. While most diabetics with bezoars will show other signs of autonomic neuropathy, the absence of such dysfunction does not exclude this possibility as demonstrated by one of our patients *(Table II)*. Since barium studies are inadequate to visualize bezoars, and as measurement of gastric emptying time does not always predict their possible presence, endoscopy is clearly needed both for diagnostic accuracy and for therapeutic benefit.

TABLE II
SOME FACTORS PREDISPOSING TO BEZOAR FORMATION

Local Causes	*Systemic Causes*
High fiber diets	Diabetes mellitus
Inadequate mastication	Scleroderma
Achlorhydria	Hypothyroidism
Prior gastric surgery	Myotonic dystrophy
Gastric ulceration/ carcinoma	Systemic medications

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Pulmonary Carcinoma

Means of Diagnosis and Treatment

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LUNG CANCER is the most common cause of death from cancer in men and will soon become so in women.[1] The incidence of lung cancer is increasing in both sexes.[2-4] In 1981, approximately 122,000 Americans developed lung cancer, and 105,000 of them died.[5, 6] If the current trend continues, 295,000 new cases will be seen in the year 2000.[7] The natural history of lung cancer is triphasic and slowly insidious. The first or pre-clinical phase usually lasts several years during which time increasing degrees of cellular atypia are found on sputum cytology. The second phase involves a progression of the atypical cytology to carcinoma in situ. The final phase is one manifested by a relatively short clinical illness at which time the disease becomes evident. At initial clinical presentation, only 20-25 per cent of patients will be candidates for surgery — the most effective mode of treatment. Almost 50 per cent will already have systemic manifestations of the disease at this time.[8, 9] This study was undertaken to review methods by which resectable lung cancer was evaluated.

Methods

Different modes of treatment and diagnostic evaluation leading to that treatment were retrospectively reviewed in 142 patients who were initially diagnosed with lung cancer and felt to be surgical candidates at Wesley Medical Center for the years 1979 to 1981. Of 142 potential surgical candidates, 102 ultimately underwent operation for intended resection. Forty were re-classified as non-operative and three patients were excluded from the study due to charting inconsistencies. Ninety-nine patients were then introduced into the study group. Of these patients, 66 were males and 33 females, a ratio of 2:1. Average age for the male group was 64 years and for the females, 66 years.

Results and Discussions

Presentation of lung cancer clinically is usually manifested in one of three ways: (1) development of symptoms, exacerbation of symptoms, or a new physical finding; (2) an abnormal shadow on routine chest x-ray in an asymptomatic patient; and (3) neoplastic cells detected in sputum of a patient whose chest x-ray appears normal. In our group, 58 per cent of surgical patients were symptomatic. Sixty-three per cent of males and 52 per cent of females were symptomatic. Presenting symptoms included: cough 50%; hemoptysis 33%; fever 21%; chest pain 14%; weight loss 12%; shortness of breath 12%; weakness and fatigue 12%; and hoarseness 7% *(Table I)*. Seventy-nine per cent of the surgical patients were smokers; 87 and 61 per cent of males and females respectively were smokers.

Reviewed here are 444 cases of pulmonary carcinoma diagnosed and initially treated at Wesley Medical Center between 1979 and 1981. It encompasses patient background and presentation, diagnostic procedures, treatment modalities, and survival information. The extent and results of pulmonary resections in those who were operative candidates (23%) are presented.

Thirty-seven per cent of the males were asymptomatic and presented with an abnormal shadow on routine chest x-ray. Forty-eight per cent of the women presented in a similar manner. A solitary mass was seen on the chest x-rays of the surgical group in 69 per cent. Twenty-three per cent had non-specific findings, including pneumonitis, atelectasis, infiltrates, and effusion. Eight per cent had combined findings of a mass in association with one of the non-specific findings *(Table II)*. The tumor was located centrally in 48 per cent of the patients in the surgical group and peripherally in 52

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TABLE I
PRESENTING SYMPTOMS

Symptom	%
Cough	50
Hemoptysis	33
Fever	21
Chest pain	14
Weight loss	12
Shortness of breath	12
Weakness/fatigue	12
Hoarseness	7

TABLE II
CHEST REONTGENOGRAM

Findings	%
Solitary mass	69
Nonspecific findings	23
Pneumonitis	
Atelectasis	
Infiltrate	
Effusion	
Combined (mass and nonspecific finding)	8

TABLE III
HISTOLOGY AT DIAGNOSIS — ENTIRE GROUP

Histology	%
Adenocarcinoma	29
Squamous cell carcinoma	30
Large cell carcinoma	4
Oat cell carcinoma	18
Carcinoma (Nos)	13
Carcinoid	2
Clinical diagnosis	4

TABLE IV
HISTOLOGY AT DIAGNOSIS — SURGICAL GROUP

Histology	%
Adenocarcinoma	48
Squamous carcinoma	38
Large cell carcinoma	3
Oat cell carcinoma	4
Carcinoma (Nos)	2
Carcinoid	5

TABLE V
MEANS OF FIRST TISSUE DIAGNOSIS

Procedure	%
Sputum	23
Bronchoscopy	
Washing	19
Brushings	9
Tissue biopsy	2
Fine needle biopsy	8
Mediastinoscopy	3
Mediastinotomy	1
Thoracotomy	34

TABLE VI
OPERATIVE PROCEDURES

Procedure	%
Pneumonectomy	13
Lobectomy	51
Bilobectomy	10
Combined lobectomy/adjacent wedge resection	4
Wedge resection	6
Unresectable	16

per cent. A central lesion was defined as any lung mass with a center that fell within a 4 cm radius of the hilum. Peripheral lesions fell outside this radius.[10]

Approximately 90-95 per cent of lung cancer arises in the bronchial or alveolar surface epithelium or from bronchial mucous glands. The four major histologic types of lung cancer include squamous cell carcinoma, adenocarcinoma (including bronchoalveolar carcinoma) small cell (oat cell) carcinoma, and large cell anaplastic carcinoma (containing undifferentiated tumors that cannot be classified as poorly differentiated adenocarcinoma or squamous carcinoma by light microscopy).[11, 12] A fifth category — carcinoma not otherwise specified (NOS) — included a diverse group of tumors, some of which could be classified only as carcinoma or bronchogenic carcinoma. Additionally, there were several in this group classified as muco-epidermoid tumors and adenoid cystic tumors.

The histology at diagnosis in the entire group of patients in this study included adenocarcinoma 29%, squamous cell carcinoma 30%, large cell carcinoma 4%, oat cell carcinoma 18%, carcinoma not otherwise specified 13%, carcinoid 2%, and clinical diagnosis only 4% *(Table III)*. The histology at diagnosis in the surgical group was made up of adenocarcinoma 48%, squamous cell carcinoma 38%,

large cell carcinoma 3%, oat cell carcinoma 4%, carcinoma not otherwise specified 2%, and carcinoids 5% *(Table IV)*.

The mode of first tissue diagnosis in the surgical group (the means by which a tissue diagnosis was first established) was found to result from sputum cytology in 23%, bronchoscopy in 30% (including washings 19%, brushings 9%, tissue biopsy 2%), fine needle biopsy in 8%, mediastinoscopy in 3%, and mediastinotomy in 1% *(Table V)*. The first means of tissue diagnosis was obtained by thoracotomy in 34%. Of this group 74% were solitary peripheral nodules.

Sputum cytology was obtained in 68 per cent of patients, and an overall positivity of 33 per cent was obtained. Eighty per cent of the centrally located adenocarcinomas were positive on sputum cytology as were 53 per cent of the squamous cell tumors. The yield for peripheral tumors on sputum cytology was less than one-third.

Bronchoscopy was performed in 87 per cent of the surgical group. It was grossly positive in 30 per cent with an additional 5 per cent showing extrinsic compression or widening of the main stem carina or segmental carina. Washings were positive in 51 per cent of the individuals in which they were obtained, brushings in 45 per cent, and tissue biopsy in 46 per cent.

Pre-thoracotomy mediastinal exploration was performed in a selective fashion and was limited to those individuals with the following criteria:

- Large (T3) central or peripheral tumors.
- Radiographic evidence of mediastinal disease (N2 disease).
- High risk patients in whom thoracotomy was to be avoided unless curative resection was highly probable and a tissue diagnosis had not been made by lesser means.
- Cases where it was highly probable that a complete resection would require pneumonectomy.
- Suspected but histologically unproven oat cell carcinoma.

In our series, 15 per cent of surgical patients underwent mediastinoscopy, and 40 per cent of these were positive. Mediastinotomy, or the Chamberlain procedure, was performed in 7 per cent of the surgical group, primarily for left upper lobe lesions, and was positive in 57 per cent of these. Low ipsilateral peritracheal intranodal metastases were not considered to contraindicate thoracotomy in patients with squamous cell carcinoma and adenocarcinoma. In these cases resection of the tumor was accompanied by adjacent mediastinal node dissection.

Fine needle aspiration biopsy was performed in 17 per cent of surgical candidates and was positive in 59 per cent of these. Fine needle aspiration was reserved for those patients where a histologic diagnosis could not be obtained by other procedures prior to potential thoracotomy to simplify the operative procedure by not requiring intraoperative biopsy. In some of these individuals the possibility of an oat cell carcinoma was entertained while in others it was felt that, because of their borderline potential for being a surgical candidate, a tissue diagnosis should be obtained if possible prior to thoracotomy.

The routine use of multi-organ scans in evaluating patients with lung cancer preoperatively is controversial. Up to 50 per cent of patients with lung cancer presentation have metastases to the mediastinum or distant organs.[8] This has been the rationale on which the use of routine scans is based. The current trend, however, is away from routine use of scans. The data in our series supports this.

Bone scans were done in 65 per cent of surgical candidates in our series and were positive in only eight. Of these, only three were positive secondary to metastatic disease, and these patients were symptomatic. Alkaline phosphatase levels were determined in 90 per cent of the surgical group and were positive in only 16. Only one of these was associated with an abnormal bone scan.

Liver scans were obtained in 76 per cent of the surgical group and were positive in only 7 per cent. Although abnormal liver function tests, which were done in 93 per cent of the surgical group, are held to be suggestive of hepatic metastases, in our series only two individuals with elevated enzymes had metastatic hepatic disease. Similarly, routine brain scans in asymptomatic individuals have extremely low yields. It is our conclusion that these three scans should be limited to patients who are symptomatic preoperatively and not be obtained routinely in asymptomatic individuals.

CT scan of the chest was obtained in only 11 per cent of operable patients in our series. Currently chest scanning should not be used to determine resectability as it does not provide a histologic diagnosis.[13] There are currently few indications for the use of CT chest scans in the evaluation of lung cancer. Possible indications, most of which are unusual, might include:

- Positive sputum cytology and negative bronchoscopy and negative radiographs.
- Known metastasis from possible lung cancer.
- The desire to exclude other chest lesions in patients with apparent solitary pulmonary metastases.

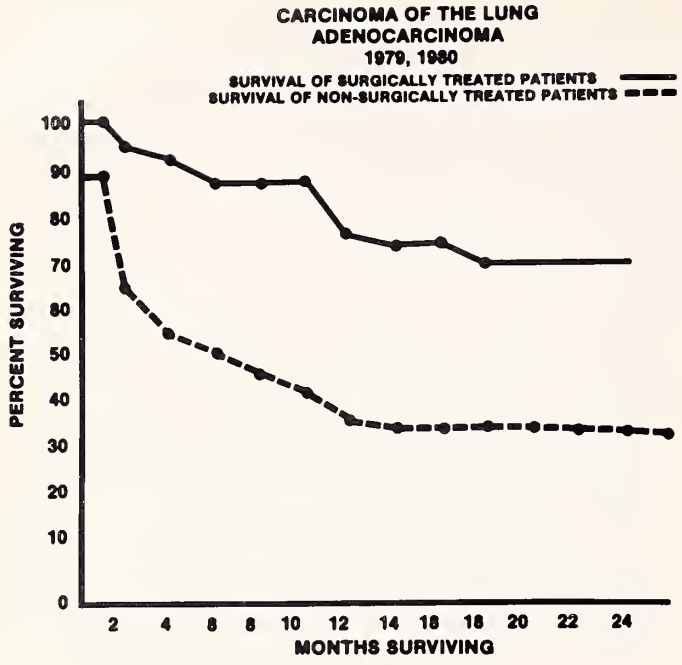


Figure 1. Comparative survival rates of surgically and nonsurgically treated patients with adenocarcinoma of the lung.

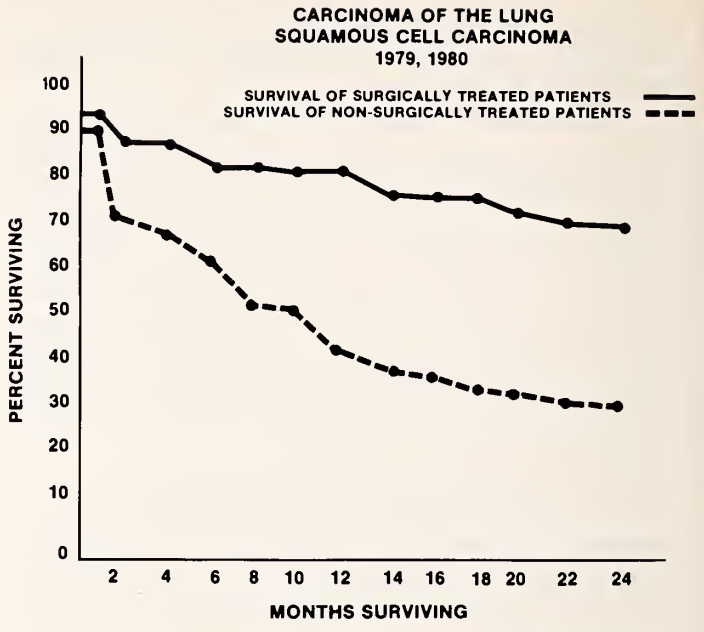


Figure 2. Comparative survival rates of surgically and nonsurgically treated patients with squamous cell carcinoma of the lung.

- The need to localize a nodule for fine needle aspiration biopsy.
- The need to identify direct extension of the tumor to the chest wall or mediastinum. (This would not necessarily indicate inoperability.)

Although not specifically analyzed and reviewed in this series, our more recent experience not included in this report does not appear to indicate a high degree of correlation between positive CT scans of the mediastinum and the presence of mediastinal metastases. A negative CT scan, on the other hand, has seemed to correlate well with the absence of mediastinal disease and in this group pre-thoracotomy mediastinal exploration may not be necessary.

The pathologic stage of the surgical group included 53 per cent Stage 1 lesions, 14 per cent Stage 2, and 33 per cent Stage 3. The American Joint Committee for Cancer Staging results[14] reporting definitions of the TMN categories was utilized in this staging classification. Pneumonectomy was performed in 13 per cent of the surgical patients, lobectomy in 51 per cent, bilobectomy in 10 per cent, lobectomy combined with adjacent wedge resection for disease crossing an inter-lobar fissure in 4 per cent, wedge resection alone 6 per cent, and 16 per cent of patients were found to be unresectable *(Table VI)*. Of the unresectable lesions, 70 per cent were central lesions in which preoperative attempts at histologic diagnosis were negative and there was no apparent mediastinal adenopathy on chest x-ray. The two year survival rate of surgically treated patients, as compared to non-surgically treated patients with adenocarcinoma of the lung *(Figure 1)* and squamous cell carcinoma of the lung *(Figure 2)* was significantly better — 35 per cent greater for adenocarcinoma and almost 40 per cent greater for squamous cell carcinoma. The overall mortality rate in the surgically treated group was 2 per cent.

Conclusions

An attempt should be made to classify patients preoperatively based on the TMN classification scheme into operable and nonoperable categories, although a standardized approach to the prethoracotomy detection of tumor and tumor spread has not yet been developed.

Reasonable attempts to obtain a histologic diagnosis prior to thoracotomy should be made in all patients. The majority of these would include and be limited to cells or tissue obtained either by sputum or bronchoscopy. If these methods are unsuccessful and there is reason to expect that mediastinal exploration would be negative, either a fine needle aspiration biopsy can be performed, or — assuming

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Pediatric Mechanical Ventilators

An Evaluation of Performance Characteristics

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MODERN VENTILATORS provide virtually all gradations of respiratory assistance, from supplementation of spontaneous breathing to total control. This broad performance spectrum can now be achieved with a number of ventilators designed specifically for use in infants and children. The principal target of pediatric ventilator design is the neonate with respiratory distress syndrome (RDS). To provide safe continuous respiratory assistance to a newborn for hours or days, a machine must be constructed with exacting specifications so that volumes and flows can be delivered with predictable accuracy. An error of 5 ml is a 20 per cent deviation from the normal tidal volume of a newborn. The precision necessary for these clinical requirements necessitates the construction of a sensitive apparatus whose performance is easily deranged. Pediatric ventilators withstand hospital abuse poorly.

As performance criteria have become more clearly defined, mechanical ventilators for adults, like automobiles, have tended to look more and more alike. This is not the case with pediatric ventilators, where marked differences in constructional design exist among currently available models. Significant differences in performance characteristics might therefore be expected. As experience with these machines is accumulated, it is evident that uniformity of performance exists only within a rather narrowly circumscribed physiologic range. When there is severe impedance to gas flow, either from loss of compliance or increased resistance of airways, the flow patterns of current makes and models begin to deviate. Discrepancies between preset values of volume and flow and those actually delivered tend to increase as positive pressure requirements are raised. This was convincingly demonstrated in a recent study by Simbruner and Gregory[1] in which volume delivery reductions, gas trapping, and prolongation of elevated intrapulmonic pressures were found to occur when compliance and resistance abnormalities were imposed. Such discrepancies are also demonstrable in adult machines,[2] and are especially prominent when high inflation pressures are required.

Neonates with RDS often require high pressures to overcome the marked restriction and obstruction due to the stiffness of the lungs. Management of the mechanical ventilation of infants with RDS, bronchopulmonary dysplasia, and pneumonia with precision and safety requires clinicians to have beforehand knowledge of the performance characteristics of the ventilators they use. This report is an evaluation of the performances of nine different pediatric ventilators, under compliance and resistance conditions prescribed by the American National Standards Institute (ANSI).[3]

Performances of nine commercially available models of pediatric mechanical ventilators were tested under varying conditions of system compliance and airway resistance. When compliances and resistances were within normal range, predicted flows and volumes were accurately delivered by all nine machines. However, variations in compliance or resistance outside of normal range resulted in volume deficits sufficient to justify a warning to clinicians to monitor flows and volumes carefully in pediatric patients with marked ventilatory impedance.

Materials and Methods

Nine different commercially available pediatric ventilators were tested with a lung model which was constructed and calibrated to provide different compliances and resistances. Measured compliances were constructed from glass bottles of 1 and 3 liter capacities. These were filled with copper wool to ensure isothermal conditions within the bottles during gas compression. The compliances selected were 3 ml/cm H_2O (C3), to simulate the lungs of a healthy

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newborn, and 1 ml/cm H_2O (C1), to simulate the lungs of a newborn with severe restrictive disease. These conditions were obtained by adding water to the glass bottles until they conformed to ANSI standards within a margin of error of ±5 per cent. The glass capacitors were connected inline to flow resistors of 50 cm H_2O/L/sec (R50) and 200 cm H_2O/L/sec (R200). These were manufactured to ANSI specifications by the Hans Rudolph Corporation, Kansas City, Missouri, and had tolerances of ±20 per cent. The R50 was intended to simulate the resistance of the normal neonatal lung, and the R200 to simulate the lung of a neonate with severe airways obstruction.

Flows were measured with a pediatric pneumotachometer (Hans Rudolph Corp.) connected to a Statham differential pressure transducer. Pressures in both the proximal airway and the lung models were measured simultaneously with Gould P23 ID pressure transducers. Volumes were computed by multiplying the compliances of the lung models by the corresponding pressures generated within them (V = CP). To check the accuracy of these calculations the volumes were also measured by integration of flows over inspiratory times, with the aid of a Honeywell Accudata 136 integrator.

Each of the nine ventilators was tested according to the protocol for waveform analysis published by ANSI.[3] The ventilators were preset to deliver 30 ml volumes 30 times per minute, with an inspiratory/expiratory time ratio of 1:2. All time-flow ventilators were operated as such — that is, there were no pressure-limited cycles. The volume ventilators were volume-cycled. All ventilators were operated with their own tubing circuits, employed according to manufacturers' instructions. The capacitors and resistors were combined in the following sequence: first C3 R50, to simulate a healthy newborn, then C3 R200, simulating marked airways obstruction, then C1 R50, simulating severe restriction, and finally C1 R200, simulating combined restriction and obstruction.

Values of all parameters measured were recorded on graph paper by a Sanborn four channel hot pen recorder. The results were considered valid only if calibrations of pressure and flow were identical before and after each test run.

Results

Quantitative data are tabulated for each machine *(Tables I-IX)*, schematics of the flow patterns are shown *(Figures 1-9)*, and noteworthy features are summarized in the following paragraphs. Individual peculiarities among the nine models were sufficiently evident so that a trained observer could easily distinguish each ventilator by its performance characteristics, unless they were closely similar in design, such as the Siemens-Elema 900B and 900C, or the Baby Bird and Baby Bird II.

All nine ventilators performed in a qualitatively similar fashion when subjected to system compliance loss or increased airway resistance. When increased airway resistance (C3 R200) was applied, there was delayed acceleration of initial inspiratory flow, plus deceleration of flow toward the end of inspiration. The flow waveform thus tended to depart from the square pattern seen with normal conditions (C3 R50), and to assume a more sinusoidal character. Application of reduced compliance (C1 R50 and C1 R200) caused the flow to decelerate throughout the inspiratory phase. Both of these effects resulted in decreased volume delivery. As impedances of both kinds were imposed, there was a net loss in peak flow as the ventilator load conditions increased. Peak airway pressures rose, with more volume compressed in the tubing circuit, and correspondingly less volume reaching the lungs. Thus there were two major sources of volume loss: attenuation of flow and increased compression in the tubing circuit.

Descriptions and performance properties of the ventilators tested are as follows:

Bourns LS-104-150 (Figure 1; Table I). This is a volume cycled, pressure limited assistor/controller. The volume and flow outputs are governed by a linear stroke piston. Under normal conditions of lung compliance and airway resistance, the flow output is constant and the flow waveform is square. Increased airway resistance causes an initial rounding off of the flow curve, indicating a loss in delivered volume. Decreased compliance does little to affect flow, but the increase in peak airway pressure will trap more volume in the tubing circuit.

Bourns BP-200 (Figure 2, Table II). This ventilator is a time/flow, pressure limited controller. With normal impedance (C3 R50) the flow waveform is square. Increased airway resistance causes the flow waveform to round off, and decreased compliance depresses the terminal portion of the flow wave (deceleration of flow). The net result is volume loss, and this is augmented by the tubing compression volume.

Bourns Bear Cub (Figure 3; Table III). This also is a time/flow, pressure limited controller. The changes in performance characteristics caused by increased flow impedance are similar to those observed with the BP-200.

Baby Bird, Baby Bird II (Figures 4, 5; Tables IV,

TABLE I
BOURNS LS104-150

	Insp time (sec)	Exp time (sec)	Insp / Exp	f (BPM)	Flowrate, machine (L/min)	Flowrate, measured (L/min)	% Flowrate decrease	Tidal Vol preset (ml)	Tidal Vol measured (ml)	% Tidal Vol decrease	PAW (cmH_2O)	Trapping in lung (ml)
C3 R50	.64	1.44	1:2.3	29	3.0	2.6	—	37	30	—	11.8	0
C3 R200	.64	1.44	1:2.3	29	3.0	2.31	11	37	26.1	13	19	3.9
C1 R50	.62	1.46	1:2.4	29	3.0	2.0	23	37	24.0	20	25	0
C1 R200	.62	1.44	1:2.3	29	3.0	1.9	27	37	23	23	29	0

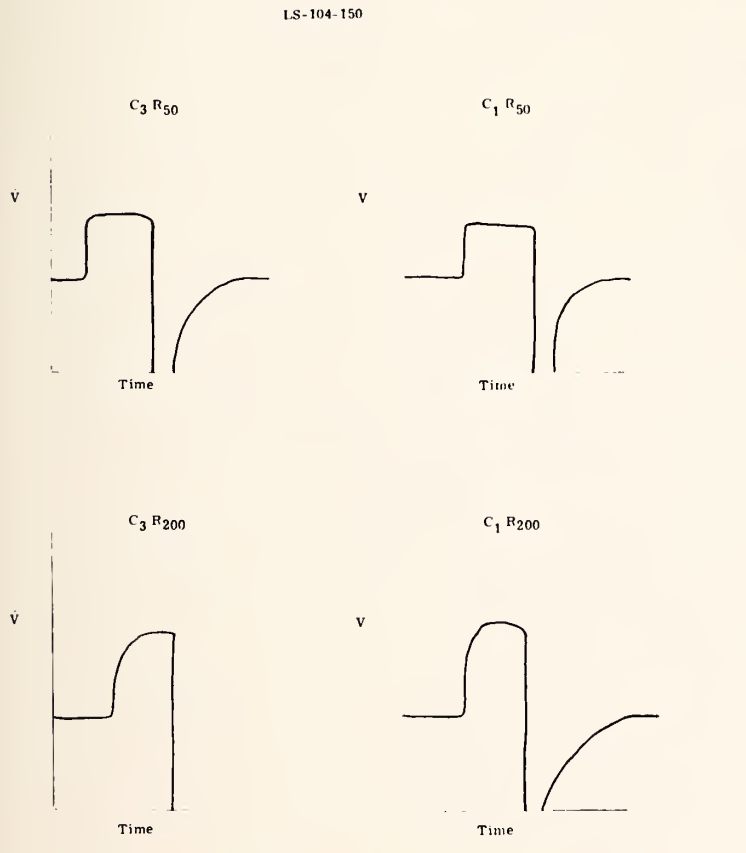


Figure 1. Bourns LS104-150.

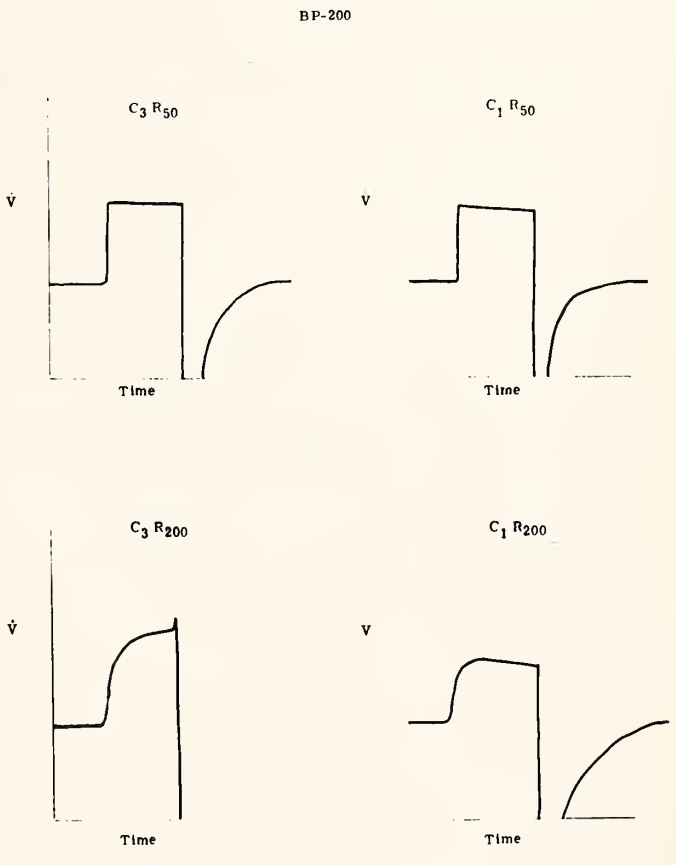


Figure 2. Bourns BP-200.

TABLE II
BOURNS BP-200

	Insp time (sec)	Exp time (sec)	Insp / Exp	f (BPM)	Flowrate, machine (L/min)	Flowrate, measured (L/min)	% Flowrate decrease	Tidal Vol preset (ml)	Tidal Vol measured (ml)	% Tidal Vol decrease	PAW (cmH_2O)	Trapping in lung (ml)
C3 R50	.72	1.36	1:1.9	29	3.5	2.2	—	N/A	30	—	12.4	0
C3 R200	.72	1.36	1:1.9	29	3.5	2.1	4.5	N/A	25.4	12	19.2	6.6
C1 R50	.72	1.36	1:1.9	29	3.5	1.65	25	N/A	24	20	25.2	0.8
C1 R200	.72	1.36	1:1.9	29	3.5	1.55	30	N/A	22.4	25	28.4	0.8

TABLE III
BOURNS BEAR CUB

	Insp time (sec)	*Exp time (sec)*	*Insp / Exp*	*f (BPM)*	*Flowrate, machine (L/min)*	*Flowrate, measured (L/min)*	*% Flowrate decrease*	*Tidal Vol preset (ml)*	*Tidal Vol measured (ml)*	*% Tidal Vol decrease*	*PAW (cmH_2O)*	*Trapping in lung (ml)*
C3 R50	.78	1.28	1:1.6	29	3.0	2.8	—	N/A	30	—	11.5	0
C3 R200	.78	1.30	1:1.7	29	3.0	2.4	14	N/A	25.5	15	17.5	4.5
C1 R50	.74	1.32	1:1.8	29	3.0	1.9	32	N/A	22	27	22.5	0
C1 R200	.76	1.32	1:1.7	29	3.0	1.8	36	N/A	20	33	24	0

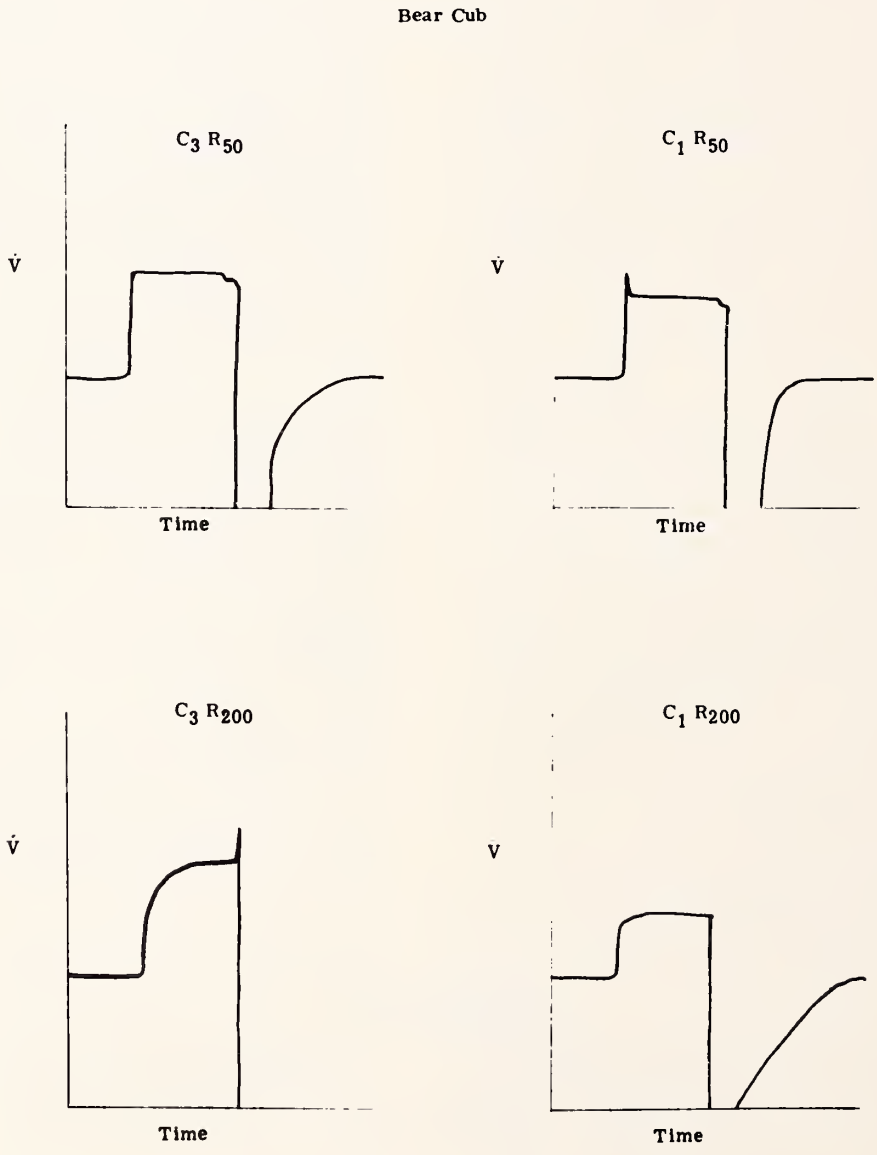


Figure 3. Bourns Bear Cub.

TABLE IV
BABY BIRD

	Insp time (sec)	*Exp time (sec)*	*Insp / Exp*	*f (BPM)*	*Flowrate, machine (L/min)*	*Flowrate, measured (L/min)*	*% Flowrate decrease*	*Tidal Vol preset (ml)*	*Tidal Vol measured (ml)*	*% Tidal Vol decrease*	*PAW (cmH_2O)*	*Trapping in lung (ml)*
C3 R50	.76	1.32	1:1.7	29	1.0	1.9	—	N/A	30	—	12	1.5
C3 R200	.76	1.32	1:1.7	29	1.0	1.8	5	N/A	25.5	15	17.5	6.0
C1 R50	.72	1.32	1:1.8	29	1.0	1.1	42	N/A	21	30	22	0
C1 R200	.72	1.32	1:1.8	29	1.0	1.05	45	N/A	19	37	24	0.5

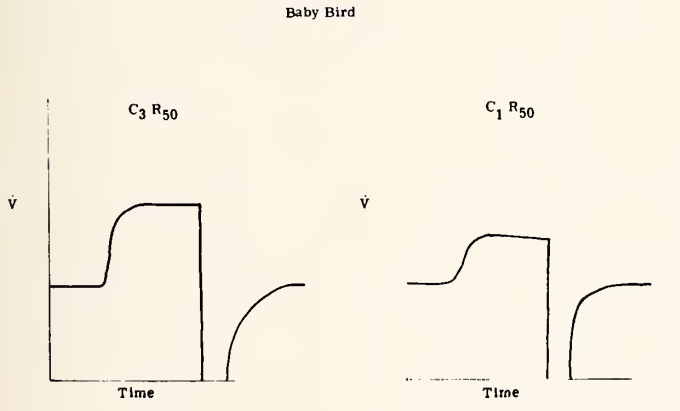


Figure 4. Baby Bird.

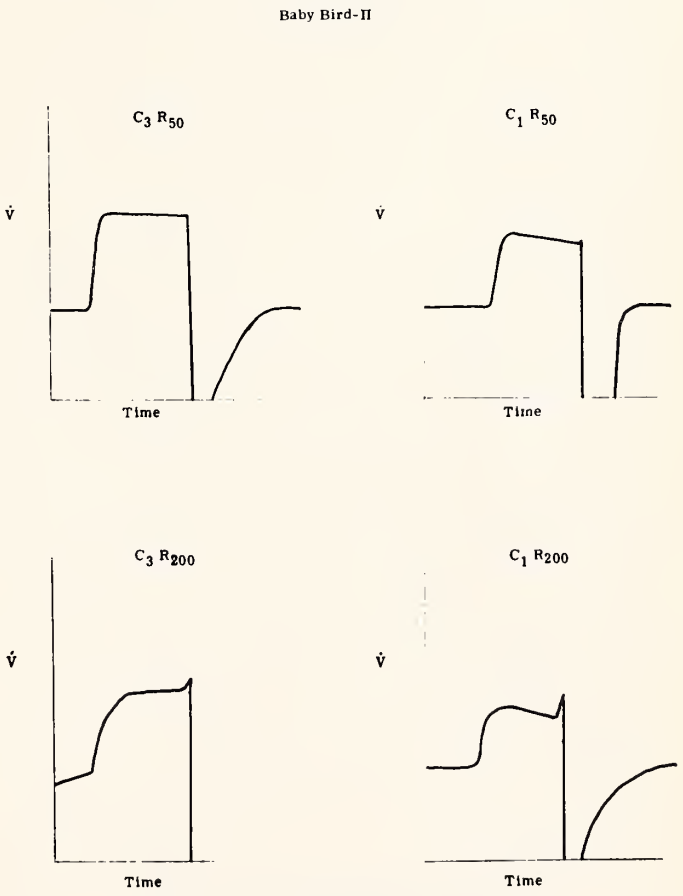


Figure 5. Baby Bird II.

TABLE V
BABY BIRD II

	Insp time (sec)	*Exp time (sec)*	*Insp / Exp*	*f (BPM)*	*Flowrate, machine (L/min)*	*Flowrate, measured (L/min)*	*% Flowrate decrease*	*Tidal Vol preset (ml)*	*Tidal Vol measured (ml)*	*% Tidal Vol decrease*	*PAW (cmH_2O)*	*Trapping in lung (ml)*
C3 R50	0.60	1.40	1:2.3	30	3.5	3.0	—	N/A	30	—	12	0
C3 R200	0.64	1.36	1:2.1	30	3.5	2.8	7	N/A	24	20	18	4.5
C1 R50	0.60	1.40	1:2.3	30	3.5	2.5	17	N/A	21	30	22	0
C1 R200	0.64	1.36	1:2.1	30	3.5	2.2	27	N/A	19	37	23	0

TABLE VI
SIEMENS SERVO 900B

	Insp time (sec)	*Exp time (sec)*	*Insp / Exp*	*f (BPM)*	*Flowrate, machine (L/min)*	*Flowrate, measured (L/min)*	*% Flowrate decrease*	*Tidal Vol preset (ml)*	*Tidal Vol measured (ml)*	*% Tidal Vol decrease*	*PAW (cmH_2O)*	*Trapping in lung (ml)*
C3 R50	.56	1.36	1:2.4	31	3.4	2.8	—	37.3	30	—	12.4	0.3
C3 R200	.58	1.36	1:2.3	31	3.4	2.6	7	37.3	29.4	2	20	5
C1 R50	.58	1.36	1:2.3	31	3.4	2.05	27	37.3	25.3	16	26	0
C1 R200	.58	1.36	1:2.3	31	3.4	2.0	29	37.3	24	20	29.6	0

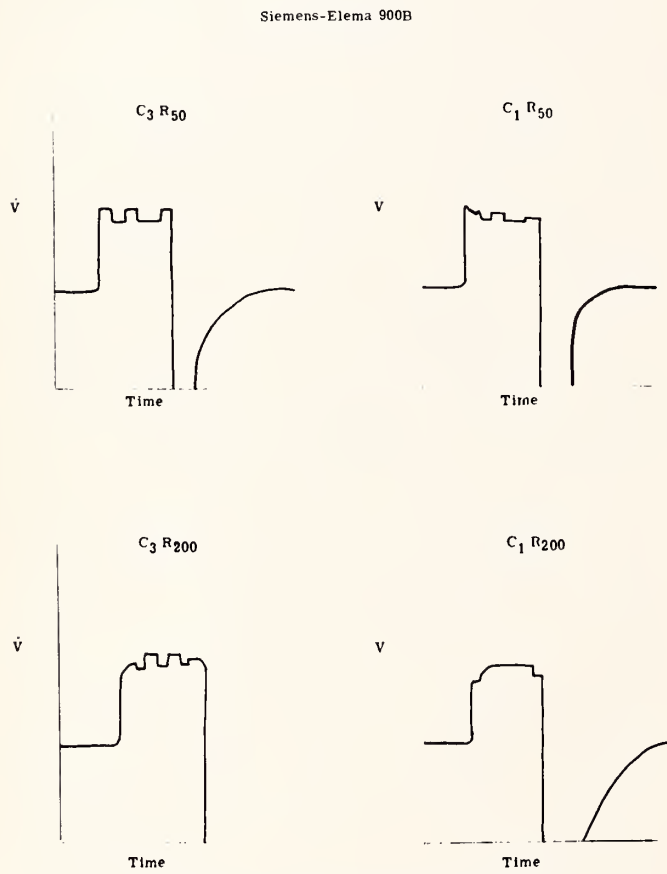


Figure 6. Siemens Servo 900B.

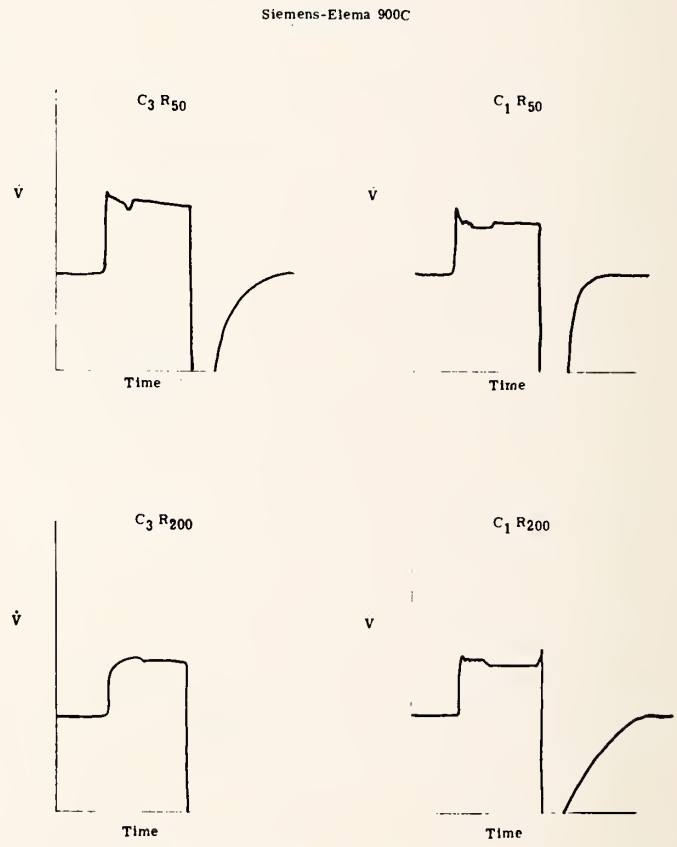


Figure 7. Siemens Servo 900C.

TABLE VII
SIEMENS SERVO 900C

	Insp time (sec)	*Exp time (sec)*	*Insp / Exp*	*f (BPM)*	*Flowrate, machine (L/min)*	*Flowrate, measured (L/min)*	*% Flowrate decrease*	*Tidal Vol preset (ml)*	*Tidal Vol measured (ml)*	*% Tidal Vol decrease*	*PAW (cmH_2O)*	*Trapping in lung (ml)*
C3 R50	.64	1.44	1:2.3	29	2.7	2.0	—	31	30	—	11.9	0
C3 R200	.64	1.44	1:2.3	29	2.7	1.2	40	31	28.8	4	19.5	3.6
C1 R50	.64	1.44	1:2.3	29	2.7	1.0	50	31	24	20	24	0
C1 R200	.64	1.44	1:2.3	29	2.7	0.8	60	31	22	27	26	0

TABLE VIII
SECHRIST IV-100B

	Insp time (sec)	*Exp time (sec)*	*Insp / Exp*	*f (BPM)*	*Flowrate, machine (L/min)*	*Flowrate, measured (L/min)*	*% Flowrate decrease*	*Tidal Vol preset (ml)*	*Tidal Vol measured (ml)*	*% Tidal Vol decrease*	*PAW (cmH_2O)*	*Trapping in lung (ml)*
C3 R50	.74	1.24	1:1.7	30	2.0	3.2	—	N/A	30	—	12.6	0
C3 R200	.74	1.24	1:1.7	30	2.0	2.2	31	N/A	24	20	18.8	6.6
C1 R50	.68	1.28	1:1.9	31	2.0	2.6	19	N/A	20	33	22.4	0
C1 R200	.74	1.24	1:1.7	30	2.0	2.0	38	N/A	19.3	36	24.9	0.4

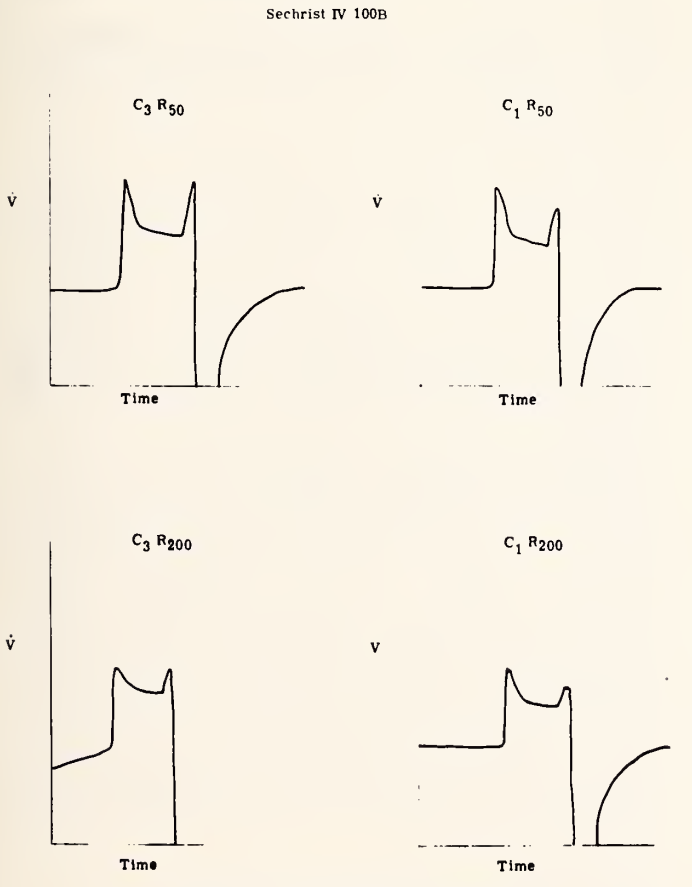


Figure 8. Sechrist IV-100B.

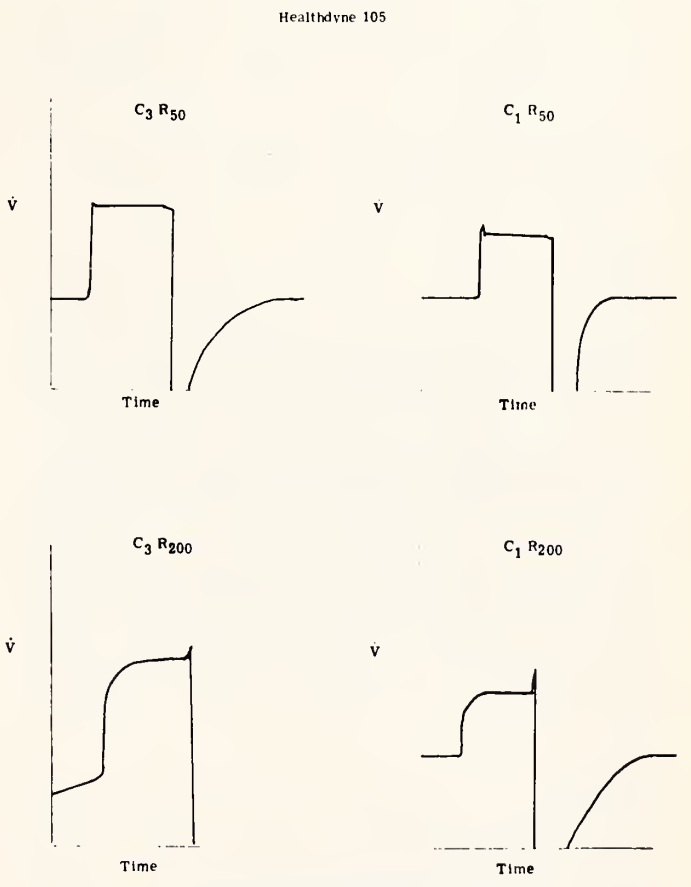


Figure 9. Healthdyne 105.

TABLE IX
HEALTHDYNE 105

	Insp time (sec)	*Exp time (sec)*	*Insp / Exp*	*f (BPM)*	*Flowrate, machine (L/min)*	*Flowrate, measured (L/min)*	*% Flowrate decrease*	*Tidal Vol preset (ml)*	*Tidal Vol measured (ml)*	*% Tidal Vol decrease*	*PAW (cmH_2O)*	*Trapping in lung (ml)*
C3 R50	.76	1.24	1:1.6	30	2.75	2.05	—	N/A	30	—	11.6	1.2
C3 R200	.76	1.24	1:1.6	30	2.75	1.95	5	N/A	26.6	11	18	6.6
C1 R50	.74	1.28	1:1.7	30	2.75	1.6	22	N/A	24	20	24	0.8
C1 R200	.72	1.28	1:1.8	30	2.75	1.5	27	N/A	22.4	25	27.2	0.8

V). These ventilators are time/flow cycled, pressure limited controllers. Their performance characteristics in this study were found to be nearly identical. Under normal load conditions, the flow wave has a slightly rounded form. This is increased on the downward slope when compliance loss is imposed. Impedance due to airway resistance causes a marked rounding off of the waveform, indicating volume loss.

Siemens-Elema 900B and 900C (Figures 6, 7; Tables VI, VII). These models are servo-operated, pressure limited, minute volume cycled assistor/controllers. Volume output is a product of integrated flow. A constant volume is theoretically maintained by electronically comparing the inspired volume with the expired volume, and adjusting the flowrate if there is a difference between the two. The flow waveform of these ventilators decelerates only slightly with compliance loss, but rounds off appreciably with increased airway resistance.

Sechrist IV-100B (Figure 8; Table VIII). This ventilator is a time/flow, pressure limited controller. The flow waveform shows an initially high peak, which then falls to a plateau and again peaks at the end of the inspiratory phase. This form stays basically the same through all four load conditions, but the flow rate is diminished with increased impedance.

Healthdyne 105 (Figure 9; Table IX). This also is a time/flow, pressure limited controller. The square flow waveform seen under normal load conditions shows slight deceleration with compliance loss and rounding off with increased airway resistance. As with all previously described machines the deceleration of flowrate is accompanied by a decrease in volume delivery.

Discussion

First generation pediatric ventilators were essentially miniaturized models of adult machines. They maintained alveolar ventilation satisfactorily in patients with essentially normal lungs. Where oxygen transport was impaired, however, they fell short of expectations. Designers of the present (''second'') generation of pediatric ventilators recognized that a number of modifications of existing adult models had to be provided in order to improve oxygenation in infants suffering from restrictive and obstructive respiratory conditions, particularly RDS.

Development of pediatric ventilators has been hampered by caveats inherited from respiratory physiology, and from experience with adult ventilators. For example, the belief was prevalent that all the gas breathed by the patient should be metered by the internal delivery system; that is, each patient breath must coincide with a machine cycle. Another assumption was that the ''natural'' breathing pattern should be closely simulated, with zero pressure at the end of expiration, no inspiratory hold or plateau, and an inspiratory/expiratory time ratio that did not exceed 1/1. Gradually all these caveats have been breached, with the result that the present models have the capability to deliver a wide variety of pressure and flow waveforms, and to provide intermittent breaths from the machine superimposed on the patient's spontaneous ventilation. Continuous positive pressure throughout the respiratory cycle has become generally applicable to most restrictive respiratory conditions encountered in infants and small children.

From these experiences during the past decade, a rather simple basic ventilator design has evolved for delivering intermittent mandatory ventilation to infants. This is a continuous flow generator that delivers a square flow wave. Inspiratory gas flow is terminated either on elapse of a preset time interval or attainment of a positive pressure plateau. These features, all or in part, are characteristic of the time/flow cycled, pressure limited controllers which comprise the majority of the current models. Of the nine ventilators tested in this study, six belong in this category.

Another mechanical approach to ventilation of infants with severe respiratory restriction is delivery of small volumes of gas at frequencies of 60-2400 cycles per minute. Successful application of this principle, collectively termed high frequency positive pressure ventilation (HFPPV),[4] has led to the appearance of a ''third'' generation of pediatric ventilators. Although the therapeutic role of HFPPV is yet undefined, it is expected that this mode of ventilation will be provided as an available option on the new machines.

Conclusions

From these studies the following conclusions may be drawn, together with suggestions for safety in employment of pediatric ventilators:

- Clinicians must be aware of changes in performance characteristics of pediatric ventilators when applied to infants with marked compliance loss or increased resistance of airways.
- Pediatric ventilators selected for purchase by hospitals should be screened according to their respective performances under ANSI-defined variations of compliance and resistance. For consistent clinical results it may be important to restrict

(Continued on page 282)



Congenital Rubella Syndrome

A Review of Development, Diagnosis, and Treatment

BRADFORD W. OLNEY, M.D., ***Shawnee Mission***

RUBELLA or "German Measles" in the postnatal period is a trivial and self-limiting disease. It would be of little concern to the medical community if it were not for the teratologenic effects it produces in the fetus of a mother infected during pregnancy. Rubella in children or adults is often so minor that many women are uncertain whether they have ever had the disease.

Man is the only natural host for the rubella virus known at this time. Rubella is predominantly a self-limited late winter and spring infection. Undoubtedly, person-to-person transmission occurs at very low levels in the general population throughout the summer and winter. Because of its ability to infect the human fetus chronically, rubella can also be maintained for long periods in an intrauterine conservatory. The infected infant then becomes a source for reintroduction of the virus after its prolonged stay *in utero*.

In most of the world sizeable epidemics occur every six to nine years with major ones at intervals ranging from 10 to 30 years. Attack rates in the open population have not been precisely defined, mainly because rubella is such a mild disease that not more than one in five to ten cases is recorded.

Rubella is spread to susceptible persons by infectious droplets from individuals with the disease. The respiratory tract is the usual portal of entry for the rubella virus in postnatal infections. The virus spreads from the nasopharynx to regional lymph nodes either by a transient viremia or by lymphatic channels. This initial lymph node involvement usually produces a lymphadenopathy which is one of the first overt signs of rubella infection. The enlargement of lymph nodes usually begins 10-16 days after exposure, and is followed by mild conjunctivitis and discrete rubelliform rash. The rash, which is not a constant feature of the primary infection, usually lasts three to five days. The viremia, which seems to be the route of prenatal infection, begins 9-14 days after exposure, and continues until about day 17 (post exposure). The virus can usually be recovered from the nasopharynx as early as seven days before and for 10-14 days after the rash appears. Arthritis is a fairly frequent finding in rubella, especially in adult females. Any number of signs of rubella may be present during a particular infection, or the disease may be so mild as to go unnoticed.

Rubella is a disease of little consequence except for the serious effects on the fetus of a woman infected during pregnancy. The infected fetus suffers a reduced rate of cellular growth and division, resulting in a variety of abnormalities related to the stage of fetal development at which the infection is contracted.

Editor's Note: Papers published in the Student Thesis series have been written by fourth year medical students at the University of Kansas School of Medicine. Those selected for publication have been recommended by the UKSM faculty.

There is a striking difference between the features of postnatally acquired rubella and congenital rubella. The postnatal infection is usually a self-limiting disease without any pronounced sequelae. In the prenatal infection, the fetus remains infected throughout intrauterine life, and for months to years after birth. The first stage of fetal infection is placental involvement, which requires a primary infection in the mother with a lengthy viremia. The circulation of the virus in the maternal blood allows access to the placenta, which can lead to chorionitis. This placental infection leads to infected emboli circulating in the fetal vascular system. Virus persists in the placenta for months but is most often cleared in late gestation so that recovery of the virus from the placenta at birth is infrequent.

Once the fetus is infected, the infection persists to birth, and virus has been recovered from the urine, feces, spinal fluid, and pharyngeal secretions of such newborns. At birth, about 90 per cent of congenital rubella syndrome infants are secreting virus, and one year later 17 per cent are still secreting. Persistence of virus in the eye and in the cerebrospinal fluid may be even more prolonged. Although only certain organ systems are classically malformed in congenital rubella, virus can be isolated at birth from practically every organ.

The mechanism of rubella teratology seems to involve two basic mechanisms. The first is the cytolytic effect of the virus, and the second is its ability to inhibit cell growth and maturation. It has been shown with *in vitro* cell cultures that the rubella virus has a relative incapacity to produce a demonstrable cytopathic effect. It seems likely that *in vivo* the virus does have some cytopathic ability, but this is not the main teratogenic mechanism. Although not the main feature of congenital rubella infections, the direct cytolytic effect has been observed in the myocardium, cochlea, and lens of the eye.

The primary effect rubella has on the developing fetus derives from reduction in the rate of cellular growth and division. This inhibition alters normal growth and development of each organ system. Although only certain organ systems are classically malformed in congenital rubella, the growth of the whole fetus seems to be retarded. This intrauterine growth retardation seems to explain the most constant feature in congenital rubella, which is low birth weight. Some studies have also shown that many organs in rubella-infected infants have a decreased number of cells.

The original relationship between maternal rubella during pregnancy and congenital defects was first described by Sir Norman Gregg in 1941. Gregg's original descriptions were concerned only with malformations of the eye, ear, and heart. Today the spectrum of congenital rubella syndrome includes numerous other defects, many not detectable at birth. The gestational age of the conceptus at the time of maternal infection is the single most influential factor on the outcome of pregnancy. Placental and fetal infection is more common when maternal infection is acquired during the first eight weeks of gestation. During this period, a placental infection rate up to 85 per cent has been documented, while demonstrable fetal infection during this same period is only about 50 per cent. Transmission to the fetus decreases sharply after the eighth week, while placental infection also decreases but less rapidly. Thus, after the eighth to tenth week, isolated placental infection is more common than fetal infection. The infection rate in infants declines from a high of 54 per cent in the first eight weeks to 34 per cent between nine and 12 weeks, and reaches a low of 10 per cent between 13 and 24 weeks. After this time chronic infection of the fetus is rare. Abortion and stillbirth result more often with fetal infections acquired during the first eight weeks. Congenital cataracts are attributed to infections in the sixth week of gestation, deafness in the ninth week, and heart defects from infections during the fifth to tenth weeks of gestation.

In general, the earlier the maternal infection occurs in the pregnancy, the more likelihood there is of congenital defects, while infections occurring after the 25th week carry a very low risk of fetal involvement. In fact, the outcome of maternal infection may be any one of the following: (1) no infection of the fetus; (2) resorption of the embryo (only in the earliest stages of gestation): (3) spontaneous abortion; (4) stillbirth; (5) infection of the placenta only; or (6) infection of both the fetus and placenta. The infected liveborn infant can have obvious multiple organ system involvement or, more commonly, no evident disease.

The most constant characteristic of congenital rubella is low birth weight for gestational age. One study showed as many as 60 per cent of affected infants were below the tenth percentile in weight, and 90 per cent were below the 50th percentile. It has also been shown that low birth weight is rarely the sole manifestation in congenital rubella, and that in general, the lower the birth weight the greater the number of congenital defects. There is also an increase in infant mortality in congenital rubella — usually associated with the increased risk of infection in low birth weight babies.

Certain conditions other than low birth weight

carry a poorer prognosis for survival, among them extreme prematurity, gross cardiac lesions, rapidly progressive hepatitis, extensive meningoencephalitis, and fulminant interstitial pneumonitis. These conditions must be carefully excluded, as the neonate may be able to compensate for them in the early period only to decompensate later. Some defects, such as interstitial pneumonitis, can be slowly progressive and cause major functional difficulties months after birth. Thus, infected infants require close scrutiny during the first six months of life, especially with regard to life-threatening conditions, and their prognosis must be guarded in these early months.

The main ocular defect in congenital rubella is cataracts, but other less common problems associated with the eye include pigmentary retinopathy, microphthalmus, glaucoma, and transient corneal haze. Two main types of cataracts are seen in congenital rubella, and either can occur unilaterally or bilaterally. The first type of cataract consists of a central dense area surrounded by a clear area between the lesion and the iris. The other type of cataract is uniformly dense, without a bordering clear zone. The cataracts of congenital rubella are usually present at birth, but may not become evident until several weeks later. One case of rubella associated cataracts did not develop until nine months after the child's birth.

Pigmentary retinopathy is a common abnormality of congenital rubella, and is one of the few defects (other than deafness) that tends to occur without other malformations. The retinopathy results from lack of pigment formation in the retina, and is of great significance in the diagnosis of congenital rubella. This retinopathy is more often unilateral, and is a nonprogressive lesion. These retinal changes are hard to detect in the presence of cataracts, but do not seem to affect visual acuity.

Other less common ocular lesions of congenital rubella are microphthalmus, glaucoma, transient corneal haze, and defects of the iris. Glaucoma occurs in about 3-5 per cent of cases of congenital rubella; early diagnosis is important because of the danger to visual acuity. Edema of the cornea from increased intraocular pressure must be differentiated from transient corneal haze, which usually resolves spontaneously. The pupils of rubella-infected infants may respond sluggishly to light, while the iris appears atrophic. This iris hypoplasia is often associated with either cataracts or microphthalmus.

Sensorineural deafness is the most common feature of congenital rubella, and often is the only malformation in a particular case. Auditory lesions are usually localized, involving the inner ear. These lesions occur most often in the stria vascularis, Reissner's membrane, and the tectorial membrane. Histopathological changes usually consist of inflammation, necrosis, and adhesions between membranes of the inner ear. Since the inflammation is an ongoing process, it can explain the late onset of deafness that occurs in some rubella-infected infants. This is why it is very important to follow children with congenital rubella who have normal hearing at birth. Hearing loss is usually bilateral and equal, but unilateral deafness can occur. It is very important to detect these cases of unilateral hearing loss because early speech impairment can result. Although sensorineural deafness is the most common defect in congenital rubella, not all defective hearing associated with rubella is caused by lesions in the inner ear. Some rubella-infected infants experience a hearing impairment where they can hear sounds, but can not comprehend them. This type of hearing defect is probably due to some sort of lesion in the cerebral cortex, and is very difficult to diagnose.

Most cardiac defects seen in congenital rubella are the acyanotic type. The most commonly seen defects are persistent ductus arteriosus and pulmonary artery stenosis. There seems to be no gross difference between these lesions and the same defects in nonrubella patients. Arterial intimal proliferation is the most common pathologic finding in the heart and great vessels of rubella-infected children. This proliferation can be so extensive as to occlude the vessel, and tends to affect only the larger arteries while leaving the smaller vessels untouched. Other less common cardiovascular lesions associated with congenital rubella are aortic and renal artery stenosis, septal defects, and tetralogy of Fallot. There have also been reported cases of neonatal myocarditis associated with intrauterine rubella. This myocarditis is the result of necrosis of the muscle fibers, and may lead to severe illness in the newborn.

The effects of intrauterine rubella on the central nervous system may be present at birth, or manifested years later. Approximately 25 per cent of rubella-infected infants show signs of central nervous system (CNS) involvement at birth, usually manifested by a full anterior fontanelle, irritability, and increased protein in the spinal fluid. As the affected infant grows, other signs such as microcephaly and mental retardation may become evident. Other infants present at birth with lethargy, hypotonia, full fontanelle, and later in life display a marked delay in motor development. Postmortem examina-

(Continued on page 270)



Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Sickle Cell Anemia

DAVID BODENSTEINER, M.D., *Kansas City, Kansas*

SICKLE CELL anemia (SCA) is an hereditary chronic hemolytic anemia which affects approximately one in every 600 Blacks born in the United States. The disease gets its name from the characteristically sickled shape of the erythrocytes, which occurs in conditions of hypoxia, acidosis, dehydration, or hypertonicity. The major manifestations of the disorder are related to vaso-occlusive events caused by these sickled cells. Most erythrocytes containing sickle hemoglobin can be sickled and unsickled by altering the oxygen content. However, even with 100 per cent oxygen, the process is irreversible in a small proportion of these cells called irreversibly sickled cells. Presumably it is these cells that initiate the obstruction to blood flow in the microcirculation. The resulting stasis causes hypoxia, and more cells sickle creating a vicious cycle.

Anniversary

This issue of the *Journal* marks the fifth anniversary of the publication of the "Current Comment" series. When Dr. H. B. Lindsley approached us regarding the use of such a series, it was something of a departure for the *Journal*, a potentially desirable service but one whose value would be measured by the choice of subjects, the quality of treatment, the timely and succinct presentations implied in the title, and over all, of course, the approval of the readers. It has been a success on all counts, and a singularly easy success for the *Journal*, as Dr. Lindsley and his committee have been entirely responsible for its sustained production.

The *Journal* extends its thanks to Dr. Lindsley and to the individual contributors for their efforts, and looks forward to a continuation of a mutually satisfying relationship.

SCA is inherited as an autosomal recessive trait. It is felt that this genetic mutation has persisted because it confers some protection against falciparum malaria. A single amino acid substitution, valine for glutamic acid at the sixth position in the beta chain, is responsible for hemoglobin S. The homozygous state, with two abnormal beta chains, results in sickle cell anemia, and the heterozygous, in sickle cell trait. Major variants that modify the disease are sickle-hemoglobin C (SC disease), sickle beta thalassemia (sickle-thal), and sickle-hereditary persistence of fetal hemoglobin (S-HPFH). Persons with sickle trait (AS) are usually asymptomatic carriers. In general SC and sickle-thal are milder than SS disease *(Table I)*. Fetal hemoglobin, because of its high oxygen affinity, inhibits sickling, especially when present in concentrations of greater than 10 per cent. Thus the S-HPFH people tend to do very well.

Diagnosis

Diagnosis of SCA is established by hemoglobin electrophoresis, which allows for the quantitation of hemoglobins S, A_2, F, and A. Patients with SCA generally have greater than 80-90 per cent HbS with the remainder A_2 and F. Other important laboratory parameters include blood count, reticulocyte count, red cell indices, and results of examination of the peripheral smear. The reticulocyte count is elevated and the white blood cell count may be elevated during the steady state. The platelet count may also be elevated — usually during or immediately following crisis. The sickle-prep merely indicates the presence or absence of HbS. Thus it will be positive in sickle trait, sickle variants, and SCA. It can be used as a screening test to determine if hemoglobin electrophoresis is needed.

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TABLE I
SICKLE CELL ANEMIA AND ITS VARIANTS

	Hemoglobin Electrophoresis	*Circulating Sickled Cells*	*Severity of Crisis*
Normal	AA	0	0
SCA[1]	SS	+ +	+ +
SC[2]	SC	±	+
S-thal[3]	S>A	±	+
S-HPFH[4]	SF	0	0
Trait[5]	A>S	0	0

1. Sickle cell anemia.
2. Sickle C disease.
3. Sickle beta thalassemia.
4. Sickle hereditary persistence of fetal hemoglobin.
5. Sickle trait.

TABLE II
COMMON ORGAN MANIFESTATIONS OF SICKLE CELL ANEMIA

Hematologic
- Hemolytic anemia
- Aplastic crisis
- Sequestration crisis
- Folate deficiency

Pulmonary
- Infection
- Infarction
- Insufficiency

Cardiac
- Congestive failure
- Cardiomegaly

Gastrointestinal
- Hepatic sludging
- Right upper quadrant syndrome
- Cholelithiasis
- Viral hepatitis
- Autosplenectomy

Skeletal
- Dactylitis
- Osteomyelitis
- Aseptic necrosis

Cerebrovascular
- Thrombotic stroke
- Hemorrhagic stroke

Genitourinary
- Hyposthenuria
- Hematuria
- Papillary necrosis
- Pyelonephritis
- Priapism
- Proteinuria

Clinical Manifestations

Virtually all body systems are affected in SCA *(Table II)*. The chronic hemolytic anemia reduces the patient's energy and lowers exercise tolerance, as well as causing secondary effects such as cardiomegaly, marrow hyperplasia, folate deficiency, and gallstone formation. The most prominent feature of vaso-occlusive events is pain. Recurrent episodes lead to ischemia and tissue damage resulting in autosplenectomy, hyposthenuria, pulmonary insufficiency, retinopathy, and strokes. Infection is a serious problem which precipitates many hospital admissions and is the most frequent cause of death. A markedly increased susceptibility to pneumococcal and hemophilus infections is a lifelong problem. Infants do well because 70-80 per cent of their hemoglobin is fetal hemoglobin. However, anemia appears by 3 months of age, and splenomegaly develops between 4-7 months. Although enlarged, the spleen becomes functionally abnormal. Once this functional asplenia occurs, the incidence of infection increases markedly, and mortality peaks during this period. Vaso-occlusive events begin in the latter half of the first year.

Sickle cell crisis, which is defined as a major disturbance in the steady state of an SCA patient, consists of two types — vaso-occlusive and anemic. Vaso-occlusive crises are far more common with severe pain from tissue necrosis the prominent feature. Fifty per cent of patients will have experienced a painful episode by 1 year of age, and the average incidence in one large series was four painful crises per year. Musculoskeletal pain is most common with abdominal pain second in frequency. Narcotic analgesics are needed for severe pain while milder episodes can be controlled with aspirin or acetaminophen with codeine.

Anemic crises are of two types — aplastic and splenic sequestration. Aplastic crises, often associated with infections, are caused by a period of reduced or arrested erythropoiesis. This phenomenon has been shown to occur in normal people, but is of little consequence, as red cell survival is normal. However since red cell survival in SCA usually ranges from 10-30 days, the hematocrit can drop 25-50 per cent following a one week aplastic period. This problem is self-limited, but blood transfusions may be needed acutely. Megaloblastic arrest can also occur — usually due to folate deficiency.

Splenic sequestration results from entrapment of large numbers of red blood cells in the spleen for a period of several hours. It is seen both in SCA and sickle variants. If sufficient blood is entrapped, massive splenic enlargement, hypovolemia, shock, and death may occur. Treatment consists of volume expanders and red blood cell transfusions. Within one to two weeks the trapped red cells are released from the spleen.

Bacterial Infection

The increased risk of bacterial infection and its causes have been reviewed by several groups. Barrett-Conner calculated the relative risk for various infections *(Table III)*. Immunologic abnormalities play a role in these increased rates. Functional asple-

TABLE III
INCREASED RISK OF PROVEN INFECTIONS IN PATIENTS WITH SCA[1]

Infection	*Increased Risk*
Pneumococcal meningitis	579
Hemophilus meningitis	116
Bacterial meningitis	309
Salmonellosis	25
Tuberculosis	8

1. Retrospective statistical comparison to general population calculated from Dade County Annual Health Report data, Dade County, Florida. (From Barrett-Conner, E. Bacterial Infection and Sickle Cell Anemia. *Medicine* 50:97-112, 1971.)

nia may be the most important, as the spleen removes particulate matter such as bacteria; it is also a site of antibody production. Other abnormalities include decreased opsonization of pneumococci, a defect in the activation of the alternate pathway of complement, and abnormal granulocyte kinetics.

The presence of bacterial infection should be assumed in all febrile patients with SCA, particularly children. Cultures of blood, urine, throat, sputum, and — if indicated — spinal fluid must be done. Broad spectrum antibiotic therapy should be initiated, and then adjusted according to the culture results. A leukocyte alkaline phosphatase score of 100 or greater is strong evidence that infection is present.

Prevention of infection is important. Pneumococcal vaccine should be given to all patients with SCA. Antibody responses are suboptimal, particularly in children, but the vaccine does seem effective. A study compared infection rates in asplenic and SCA individuals and found eight pneumococcal infections in the control group, all due to types found in the vaccine, and none in the vaccinated group. The role of prophylactic antibiotics has not been established. Finally, parents and patients should be aware of the signs of infection and seek medical care early.

Organ Involvement

Acute pulmonary events occur in nearly 50 per cent of patients and are a major cause of morbidity and mortality. The manifestations include pulmonary infiltrates, pulmonary emboli, fever, leukocytosis, chest pain, and respiratory distress. They may be due either to infection, infarction, or both. Pneumococcus is the most common infectious organism, but the incidence of hemophilus, staphylococcus, and mycoplasma is increasing. Vaso-occlusive events are also common. They may occur as a primary event, or as a complication of pneumonia. Chest pain without hypoxia is usually due to infarcts of the rib cage. The end result of recurrent pulmonary events is progressive pulmonary insufficiency.

The effects on the heart are due to chronic anemia. Myocardial infarctions are rare. Cardiac dilatation and ejection murmurs are secondary to the hyperdynamic circulation. Severe heart failure may develop in adults, and pulmonary insufficiency may exacerbate this problem.

Hepatobiliary disease in SCA patients can be divided into four areas: acute hepatic sludging, acute right upper quadrant (RUQ) syndrome, cholelithiasis, and viral hepatitis. In acute hepatic sludging, patients have malaise, mild RUQ pain, palpable tender livers, and hyperbilirubinemia to 20 mg/dl or greater. Liver enzymes and alkaline phosphatase are mildly elevated. The clinical course is short — usually less than one week. Liver biopsy shows sludging of the sickled cells in the sinusoids, bile stasis, and no inflammatory component. The acute RUQ syndrome consists of hyperbilirubinuria and RUQ pain. Laboratory tests are quite variable. Gallstones are a major cause of the syndrome, but the symptoms persist in some patients postcholecystectomy, and occur in others who do not have gallstones. Cholelithiasis occurs in more than 50 per cent of adults with SCA. Stones are usually pigmented because of chronic hemolysis. Cholecystectomy is recommended in symptomatic individuals, but controversy exists regarding the asymptomatic. The incidence of viral hepatitis may be higher in SCA because of frequent transfusions. Little is known concerning chronic liver disease. Iron overload is commonly seen in patients who have had many transfusions.

Skeletal abnormalities include biconcave cupping of vertebral bodies, loss of trabeculae, thinning of the outer table of the skull, and expansion of the maxillary bones — all due to marrow hyperplasia. Bones are a common site of painful crisis. Dactylitis, usually the initial painful episode seen in children, consists of fever, leukocytosis, and symmetrical swelling of the hands and feet. This disorder is self-limited and is rarely seen in children beyond the age of 7 years. Bone pain, particularly in the long bones, precipitates many hospital admissions. It is usually due to vaso-occlusion and infarction, rather than infection. However, the incidence of osteomyelitis is markedly increased in SCA, with salmonella and staphylococcus the usual organisms. Aseptic necrosis of the femoral and humeral heads from recurrent infarcts occurs more frequently in SC

disease. Synovial thickening and non-infective effusions are also seen.

Cerebrovascular accidents are the most dramatic and debilitating complications of SCA. The incidence in one large series was 6 per cent, and the recurrence rate was an extraordinarily high 67 per cent, most occurring within three years. Good neurologic recovery usually follows infarcts, but permanent paralysis can occur. Retinopathy is seen more frequently in SC disease than SCA. Arteriolar occlusion results in vitreous hemorrhage and retinal detachment. Laser photocoagulation has been used with some success.

Disorders seen in the genitourinary system include hyposthenuria, hematuria, papillary necrosis, pyelonephritis, priapism, and rarely nephritis or nephrosis. Hyposthenuria — the inability to concentrate urine normally — is the most consistent feature of sickle nephropathy. It is due to microinfarction of the renal medulla. Papillary necrosis, likewise due to renal medullary infarction, is seen in more than 50 per cent of SCA patients at some time. This, as well as more subtle papillary changes, results in hematuria. Pyelonephritis and cystitis are common problems. Priapism — the spontaneous and painful engorgement of the penis — occurs in 5 per cent of male patients; surgical treatment is required if parenteral fluids and transfusions fail. Proteinuria is a common finding, but it is rarely in the nephrotic range. Hyposthenuria and hematuria can also occur in sickle trait.

TABLE IV
ACUTE MANAGEMENT OF SEVERE SICKLE CELL CRISIS

Analgesia	Demerol 50-100 mg IM or morphine 5-10 mg IM every 2-4 hrs
Fluids	150-200 cc/hr
Oxygen	2-4 liters/min, only helpful when underlying pulmonary problem present
Antibiotics	When evidence of infection exists, penicillins are the drugs of choice. Gram-negative coverage also indicated empirically.
Laboratory Studies	Hemoglobin — 6 gm/dl usually adequate. White blood cell count — elevated even in the steady state Reticulocyte count — if low consider aplastic crisis Urinalysis Chest x-ray — infiltrate indicates infection or infarction
Transfusions	Must be tailored for each patient. Usually not needed when hemoglobin greater than 6 gm/dl

Pregnancy

The risks of pregnancy are significant for the mother and the child. Spontaneous abortions, stillbirths, and neonatal deaths range from 10 to 30 per cent each in large series. Maternal deaths are uncommon, but significant perinatal morbidity occurs. Good prenatal care and close observation are very important. Folic acid and transfusion therapy, to maintain a hemoglobin greater than 10 gm/dl, is indicated, especially in the third trimester.

Contraception is a controversial area. Tubal ligation is the safest and most effective; however it is not an option for women who want to give birth later. A barrier method — either diaphragm or condom — is safer but less effective. Although controlled studies do not exist, it is generally believed that birth control pills carry a significant risk of enhanced clotting in areas of vascular stasis in SCA. Low estrogen containing pills are used by some patients, but high estrogen pills appear to be contraindicated. Medroxyprogesterone acetate (Depo-Provera) is used in other countries but currently is not approved for this use by the FDA.

Treatment

General measures for sickle crisis include oxygen, fluids, analgesics, blood transfusions if indicated, and antibiotics when infection is suspected *(Table IV)*. Folic acid replacement is indicated in all patients. A hypertransfusion program reducing the HbS to less than 30 per cent has benefitted patients with central nervous system complications, priapism, leg ulcers, and pulmonary and hepatic failure. Transfusions are also of benefit preoperatively and during pregnancy.

Newer treatment modalities are being evaluated. Anti-sickling agents in general have proved too toxic. Extracorporeal treatment reduces the toxicity, but the cost of a large scale program would be prohibitive. Induced hyponatremia reduces frequency and severity of crises, but the complications of seizures and mental confusion limit its usefulness.

A search is currently underway for agents that prevent hemoglobin switching — the process that turns off production of HbF and turns on production of adult hemoglobin. Maintaining high levels of HbF would alleviate many complications of SCA. The chemotherapeutic agent 5-azacytidine has increased HbF production in two patients with thalassemia and in two with SCA. However toxicity again

is a limiting feature. The development of a suitable nontoxic agent would permit effective treatment of this chronic debilitating disease.

Summary

In summary, sickle cell anemia is an hereditary hemolytic anemia which affects virtually every body system. Tissue damage caused by vaso-occlusion results in recurrent paintul episodes and progressive organ dysfunction. Unfortunately, no effective treatment is currently available except supportive care.

Self-Assessment Questions

1. The diagnosis of sickle cell anemia is established by the sickle-prep. (T or F)
2. The most prominent feature of vaso-occlusive events is pain. (T or F)
3. The incidence of pneumococcal infection is markedly increased in sickle cell disease. (T or F)
4. Hyposthenuria and hematuria occur in patients with sickle cell trait. (T or F)
5. The pneumococcal vaccine is of no value in sickle cell anemia. (T or F)

(Answers on page 285)

Congenital Rubella Syndrome

(Continued from page 265)

tions in children with CNS involvement showed vasculitis, ischemic necrosis of the cerebrum, and infiltration of the meninges with lymphocytes and large mononuclear cells.

One study of congenital rubella children with CNS involvement by Desmond *et al.* showed that in the 3-24 month age group the predominant CNS findings were motor delay (66%), disturbances of tone and reflexes (69%), difficulties with feeding (48%), and abnormal clinical behavior (45%). Delays in motor development were associated with hypotonia of the shoulder girdle and trunk, and either hypertonus or hypotonus of the lower extremities. In the same study, the patients were reevaluated when they reached the 3-7 year age group, and a changing neurological pattern was found. There was a decrease in the abnormalities of tone along with an increase in poor balance, uncoordination, and behavioral manifestations. The behavioral problems that showed up in many of these congenital rubella children included short attention span, impulsivity, emotional lability, and hyperkinesis. When these same children reached the 9-12 year age group, the most prominent findings were proximal muscle weakness, with deficiencies in balance, and abnormalities of gait.

Another study showed that while the percentage of rubella-infected children who were thought to be retarded decreased with age, neurotic and behavioral problems increased with age. It has been suggested that with increasing social expectations in the school age period, neurological damage present from birth may become manifested as behavioral problems. The psychiatric diagnosis of these children included reactive behavior disorders, neurotic behavior disorders, mental retardation, and autism.

Other common signs of congenital rubella that can be present at birth are hematologic changes, hepatosplenomegaly, and osteopathy. The most common hematologic problem is thrombocytopenia. The platelet count varies from 10,000 to 100,000, and purpura is a common manifestation of this condition. The purpura may be present at birth, but usually appears within the first 48 hours. The thrombocytopenia purpura of rubella often resolves without specific therapy in three to four weeks. Enlargement of both the liver and spleen is a common finding at birth in disseminated rubella. Hepatosplenomegaly may be associated with jaundice, usually within the first 24 hours. The most striking bony change in congenital rubella is an alteration in the trabecular pattern of the metaphyses of long bones, usually the distal femur and proximal tibia.

Late-onset diseases that are occasionally linked to congenital rubella are growth hormone deficiency, diabetes mellitus, and hypothyroidism. Many patients with congenital rubella remain short in stature, and there have been a few reported cases where this has been linked to a deficiency in growth hormone. Research on cases of diabetes mellitus in rubella infected children has suggested that persistent viral infection of the pancreas might be the cause. It is also possible that an immunopathological process might be the cause of late-onset diabetes in congenital rubella.

In summary, when congenital rubella involves the classical signs of cataracts or deafness, it is not hard to diagnose. It is the congenital rubella infants who present with only one manifestation or perhaps just delayed motor development who are difficult to diagnose. It is also a challenge to the physician to try to predict what problems a rubella child may develop and diagnose, and to treat them early.

A bibliography is available from the author.



Drug INFORMATION

JOYCE GENERALI, R.Ph., ***Editor***

Indomethacin-Furosemide Drug Interaction

Question: Are there reports of drug interaction involving indomethacin and furosemide?

Answer: There are several reports of such an interaction.[1-6] In 1981, Allan reported the case of a 63-year-old man treated for gout with indomethacin concurrently with furosemide and other diuretics as part of his treatment for congestive heart failure (CHF). The patient was admitted with severe CHF, and pulmonary and peripheral edema. His furosemide dosage was increased to 750 mg/day with little improvement. On the day following discontinuance of indomethacin, a brisk diuresis was noted. The patient was discharged with CHF under control and without edema.

In controlled studies, other investigators have demonstrated this interaction in healthy volunteers.[2-6] In one study of eight healthy volunteers, Brater[5] found a 20 mg dose of furosemide produced an average urine output of 2,060 ml and sodium excretion of 182 mEq during the four hours following the dose. When indomethacin was administered prior to the furosemide dose, urine volume decreased to an average of 1,525 ml and sodium excretion to 132 mEq during the four hours following the furosemide dose.

Mechanism

The mechanism(s) involved in this interaction is not well understood. Most investigators support the theory that indomethacin, as a prostaglandin inhibitor, reduces the diuretic and natriuretic response to furosemide which is partly mediated by prostaglandins.[3, 4, 7] Others suggest that indomethacin and furosemide, both weak acids, compete for active secretion into the renal tubule thus preventing furosemide from reaching its site of action.[3, 6]

Conclusion

The diuretic and natriuretic response to furosemide is reduced by concurrent administration of prostaglandin inhibitors such as non-steroidal anti-inflammatory drugs (NSAID).[1-6] Pain relief afforded by indomethacin may also be reduced when administered concurrently with furosemide.[8] It is possible that this interaction is dose dependent,[5] although case reports in which the furosemide dose was increased substantially produced little to no additional diuretic response.[1, 9] Other NSAIDs reported to reduce the response to furosemide include ibuprofen and naproxen.[2, 9] This interaction is especially significant to the CHF, hypertensive, or renal compromised patient. If this interaction is suspected, the NSAID may need to be discontinued before the diuretic response to furosemide can be achieved.

Submitted by Kathleen L. Wheeler, R.Ph. and Bruce Scott, R.Ph.

References are available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

The Drug Information Service is maintained by the Department of Pharmacy at the K.U. Medical Center to promote rational drug therapy. The Service is staffed by clinically oriented pharmacists with access to accurate, current, and unbiased information. Information is available free of charge to any health care practitioner involved with patient care. Contact:

Drug Information Service
3930 Cambridge
Kansas City, KS 66103

Telephone (any time): 913-588-2328

The President's Message



Dear Colleague:

The Kansas Medical Society has completed another successful year. Kermit Wedel has done an excellent job as president; he has been a steady hand at the helm in a troubled sea.

While we live in turbulent times, now is not the time for despair, panic, or frustration. Organized medicine will continue to increase its vocabulary, with such terms as MEDIKAN, HMO, PPO, DRG, TEFRA, RCE, HCFA, and many others invented daily. We must seek to understand all of the changes that occur, so as to be able to evaluate them and respond appropriately. Rather than be a reactive body, however, our organization must contribute to the decisions and be a part of the solutions.

Ours is the era of change. We will see changes in virtually all aspects of medicine, including the actual delivery of medical services; in the area of professional liability; the numbers and scope of practice of physician extenders, and many more. Therefore, it is now more important than ever that physicians become actively involved in supporting and guiding their Medical Society, which is the only organization truly dedicated to safeguarding the quality health care of the people.

With your individual and collective help we will succeed.

Jeannie A. Gleason M.D.

President



Pressure Points

Periodically we are constrained to warn the unsuspecting who venture into these columns that the offering may seem repetitious of earlier comments. It seems so to us, but for us it represents one of the penalties for habitually talking to one's self plus a habit of waking up at an hour too early to get up but too late to settle back for comfortable sleep. The recurrent theme which plagues us is the nature of the changes that seem the incessant accompaniment of medical practice — whether they are substantial alterations in character and purpose or only superficial shifts in mood and lighting.

The current attack was precipitated by a point of minor concern (as such releases often are). *Discover,* the representative of Time, Inc. in the world of science, is continuing to be irked by the policies of the *New England Journal of Medicine,* contending that they are depriving physicians (and consequently their patients) of needed information by their determination to reject material that has been published elsewhere and by the (to *Discover*) unconscionably long delay in bringing the material to publication. The *NEJM* has not shown any signs of being cowed by *Discover* but it can be noted incidentally that the *Journal of the American Medical Association* has, in a glow of self-commendation, published a manifesto regarding *its* policies which include a determination to move certain favored contributions to the head of the line as well as "not adhering to any arbitrary Talbott-Ingelfinger-Gelman rule," which should merit the *Discover* Good Conduct Medal.

As we have suggested, the matter in itself may not be of great importance. What is important is that it is another manifestation of the democratization of medical practice. By "democratization," we do not intend to demean the egalitarian virtues of the political system but to cite those increasing efforts to impose upon the profession forms of conduct and methods of function that are not compatible with the fundamental intent of the discipline's philosophies. There are enough in-born hazards within the execution of proper medical service without contending with the sophistries of extramural opportunists. The voices of criticism and correction have come out in full cry against medicine in recent decades. One might be puzzled by this since the same era has brought remarkable progress in the management of illness and injury but perhaps it is not so puzzling if one keeps in mind that those advances have, in themselves, burdened medicine with an expectation of perfect performance and unrelenting success — preferably without risk and at discount prices.

There is tremendous ambivalence toward the medical profession: it is praised not infrequently for obvious miracles and condemned at least as frequently if it adopts secular ways even as society is forcing them on us. This determination to fit the medical foot into the shoe of society's design has constituted the century's prime change in the profession — even more than the intrinsic medical advances since these advances have become the implements of the social efforts. This socialization that has come to medicine may not be quite of the form that so disturbed our antecedents but has done most of what they feared and some things they didn't think about.

This ambivalence has stemmed not only from the nature of professional successes but the belief of some that the profession has been deficient in resolving the social dilemmas its successes have produced. It is not that medical practice in the past has intentionally perpetrated the exclusion of any segment of society from its concern on economic or any other grounds — the over all record needs no defense — and society itself has dictated the levels of existence and privilege. But a century of military and economic and social upheaval has provided a fertile ground for broad changes, and the basic theme has been that all should be provided for all, whatever the category. By its nature, medicine was the ideal place for classlessness to be implemented. All that was required was a funding process which, in its doing,

gave to the public the assurance that *its* role had been fulfilled and any failure of the plan would be traced to the providers. But it is not our intention to inveigh against the resulting conditions. It is the question of the effect of the system upon the providers that concerns us, the changes it will induce in them — already *has* induced — as well as alterations in the basic professional and ethical concepts of the profession itself.

One of the more obvious and potent changes that has affected medical practice is the litigious climate that surrounds it. True, there is scarcely any area of service or production that has escaped the sweep of increasing liability but none has been affected more extensively than medicine. Besides the more obvious and direct stresses, it has spawned numerous incursions into the intimacies of medical service: exhaustively informed consent, the dubious benefits of second opinions, actual and threatened losses of confidentiality as third parties are admitted to the records, defensive medicine. At the moment, the possibility of any material shift away from the present condition is at least unlikely — it is more certain that the demands it imposes on the physician will be incorporated into the practice regardless of any cost-benefit ratio.

In another direction, there is no hotter item in the medical arena today than cost containment. It has promoted a bewildering variety of systems of controls (read changes) over medical service, all of them equipped with their own acronyms and claiming to be the best and highest answer to keeping medical costs in line — and no one really knows how they will work out in practice since they attempt to regulate many disparate factors — not excluding human nature. One is reminded that it hasn't been too long since "contract" physicians were anathema in the profession and that corporations were prohibited — legally and philosophically — from practicing medicine. (But then we are reminded too of another of our favorite themes — the role of words in shaping and developing our attitudes). In passing, it should be noted that the payment programs for the socially designated medical services have been a questionable "bargain" for all concerned. Much has been made of the few instances of blatant and fraudulent actions by physicians with obvious damage by association to the profession and colleagues. But the complexities of payment — both extent and method — have invited — even demanded — of honest physicians the unnecessary (medically, not bureaucratically) and redundant provision of services with a staggering final bill which proves the fiscal impossibility of the intent — with physicians, because of their position, getting the major blame.

In a little different direction, we may ask what is happening to the vaunted physician-patient relationship. With its presumed focus on the actual contact between these two principals as the clearest exercise of medical function, it may be considered to be in better shape than most of the other elements of the medical scene. But this ignores the obvious alterations in medical usage today. A more mobile population lacks the anchoring effect of a medical advisor in a firm home situation. Beyond the immediate necessities, emergency room and primary care centers thrive on turnover, not involvement with the patient in a role of extensive support. Established offices, whether providing specialized or broad range care, are increasingly staffed by multiple individuals on call at given times with the result that patients get services dictated by a professional but not personal connection. The physician-patient connection is embodied in the chart, not the interpersonal relationship.

So the list of actual and potential changes could go on at great length. No discipline loves to claim its connections with its ancient heritage more than medicine, and the question is inescapable: have we come through centuries of social and intellectual and economic and political upheaval encased in some immutable mold of professional principles and virtues (always the virtues, never the vices)? The records of our forebears were written in forms that have long passed from usage and have required the interpretation of scholars — with innumerable possibilities for varying degrees of change in emphasis and varying interpretation as they have passed through differing cultures, now to be applied in a world of medical function undreamed of in those formative times. Are we really capable of recognizing changes of a truly profound and character-altering nature?

We attempt to perpetuate our conception of those principles in oaths and formal declarations. If one wishes to frame one's motivations in such a manner, all well and good. But the declarations do not make the principles. Those are invested in the individual who professes them. "Professes." That is the key note in this essentially personal expression of belief.

Although the social storms envelop the effort in an apparent commercialism and acquiescence to temporal demands, it is within the province of the individual practitioners — and the sum of their combined efforts — to retain the principles of profession and reject the role of trade. As long as this is true, we need not fear the changes.

Well, we warned you. — *D.E.G.*

KMS Obituaries

Myrtle S. Carney, M.D., 95, Fort Worth, Texas, Creighton University School of Medicine 1912, date of death unknown.

Charles G. Foster, M.D., 62, Topeka, Jefferson Medical College (Philadelphia) 1947, died April 1, 1983.

Rene M. Gouldner, M.D., 94, Wichita, Columbia University School of Medicine 1918, died March 24, 1983.

Peter E. Hiebert, M.D., 79, Shawnee Mission, The University of Kansas School of Medicine 1934, died February 2, 1983.

Howard U. Kennedy, M.D., 64, Topeka, The University of Arkansas School of Medicine 1944, died November 14, 1982.

Henry L. Regier, M.D., 101, Kansas City, The University of Kansas School of Medicine 1907, died October 1982.

Frank A. Rieke, M.D., 66, Shawnee Mission, The University of Kansas School of Medicine 1941, died February 23, 1983.

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—G. Boss, MD et al

TOPHI, CALCULI, RENAL DISEASE

"... (1) patients with extensive tophaceous disease...; (2) patients with a history of renal calculi... since a uricosuric drug may exacerbate renal stone disease; and (3) patients with significant renal disease... who are unlikely to respond to a uricosuric drug."[2]

—Edward W. Holmes, Jr, MD

For information on adverse reactions, warnings, etc, please see brief summary of prescribing information below.

ZYLOPRIM® (allopurinol)
100 and 300 mg Scored Tablets

INDICATIONS AND USE: This is not an innocuous drug and strict attention should be given to the indications for its use. Pending further investigation, its use in other hyperuricemic states is not indicated at this time.
Zyloprim® (allopurinol) is intended for:
1. treatment of gout, either primary, or secondary to the hyperuricemia associated with blood dyscrasias and their therapy;
2. treatment of primary or secondary uric acid nephropathy, with or without accompanying symptoms of gout;
3. treatment of patients with recurrent uric acid stone formation;
4. prophylactic treatment to prevent tissue urate deposition, renal calculi, or uric acid nephropathy in patients with leukemias, lymphomas and malignancies who are receiving cancer chemotherapy with its resultant elevating effect on serum uric acid levels.

CONTRAINDICATIONS: Use in children with the exception of those with hyperuricemia secondary to malignancy. The drug should not be employed in nursing mothers.
Patients who have developed a severe reaction to Zyloprim should not be restarted on the drug.
WARNINGS: ZYLOPRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION.
In some instances a skin rash may be followed by more severe hypersensitivity reactions such as exfoliative, urticarial and purpuric lesions as well as Stevens-Johnson syndrome (erythema multiforme) and very rarely a generalized vasculitis which may lead to irreversible hepatotoxicity and death.
A few cases of reversible clinical hepatotoxicity have been noted and in some patients asymptomatic rises in serum alkaline phosphatase or serum transaminase have been observed. Accordingly, periodic liver function tests should be performed during the early stages of therapy, particularly in patients with pre-existing liver disease.
Patients should be alerted to the need for due precautions when engaging in activities where alertness is mandatory.
Occasional cases of hypersensitivity have been reported in patients with renal compromise receiving thiazides and Zyloprim concurrently. For this reason, in this clinical setting, such combination should be administered with caution.
In patients receiving Purinethol® (mercaptopurine) or Imuran® (azathioprine), the concomitant administration of 300-600 mg of Zyloprim per day will require a reduction in dose to approximately one-third to one-fourth of the usual dose of mercaptopurine or azathioprine. Subsequent adjustment of doses of Purinethol or Imuran should be made on the basis of therapeutic response and any toxic effects.
Usage in Pregnancy and Women of Childbearing Age:
Zyloprim should be used in pregnant women or women of childbearing age only if the potential benefits to the patient are weighed against the possible risk to the fetus.
PRECAUTIONS: Some investigators have reported an increase in acute attacks of gout during the early stages of allopurinol administration, even when normal or subnormal serum uric acid levels have been attained.
It has been reported that allopurinol prolongs the half-life of the anticoagulant, dicumarol. This interaction should be kept in mind when allopurinol is given to patients already on anticoagulant therapy, and the coagulation time should be reassessed.
A fluid intake sufficient to yield a daily urinary output of at least 2 liters and the maintenance of a neutral or, preferably, slightly alkaline urine are desirable to (1) avoid the theoretic possibility of formation of xanthine calculi under the influence of Zyloprim therapy and (2) help prevent renal precipitation of urates in patients receiving concomitant uricosuric agents.
Patients with impaired renal function require less drug and should be carefully

"The most important therapeutic measure is the administration of a drug which will block urate synthesis. The agent available at present is allopurinol (Zyloprim . . .) which is very effective and of low toxicity."[3]

—Alfred Jay Bollet, MD

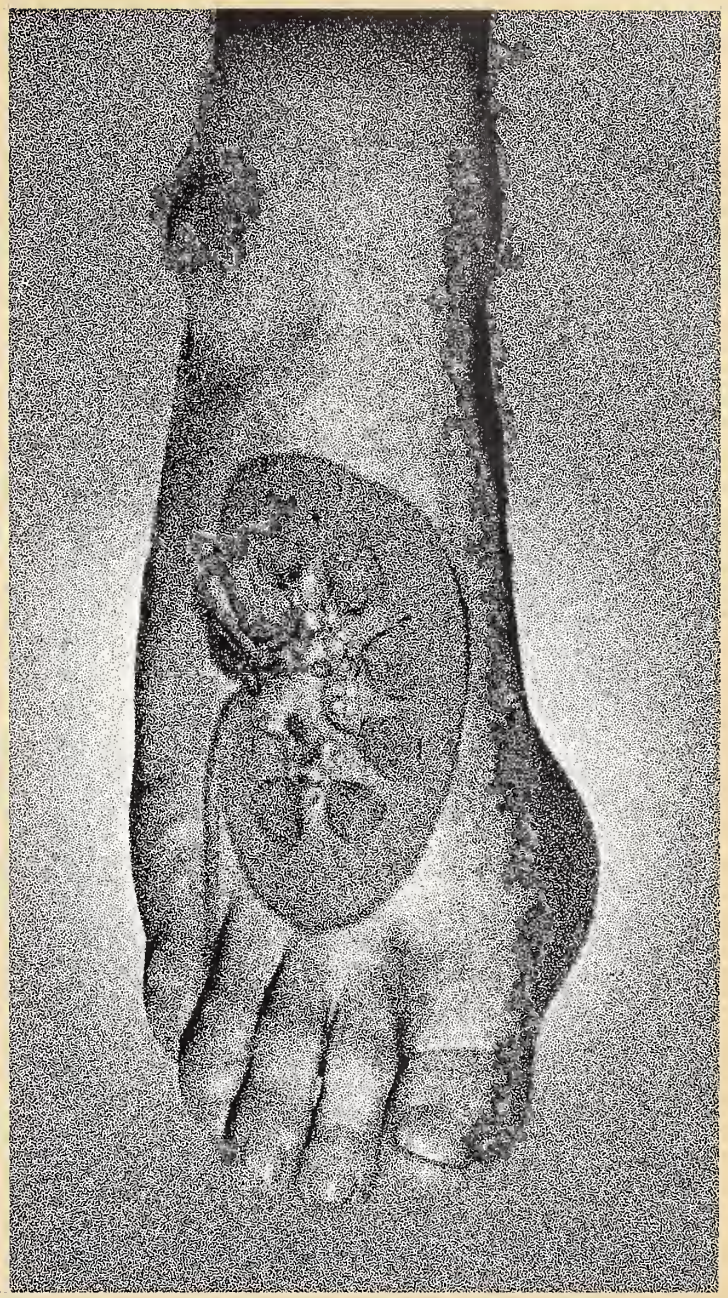
". . . allopurinol treatment appears to retard the progression of renal dysfunction."[4]

—T. Gibson, MD et al

LOW INCIDENCE OF TOXICITY

"Clinical experience with allopurinol suggests that most patients tolerate this drug well—a finding strongly supported by our data. Undesired or unintended effects of therapy were reported in only 1.8% of 1835 consecutive recipients."[5]

—G. T. McInnes, MD



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observed during the early stages of Zyloprim administration and the drug withdrawn if increased abnormalities in renal function appear.

In patients with severely impaired renal function, or decreased urate clearance, the half-life or oxipurinol in the plasma is greatly prolonged. Therefore, a dose of 100 mg per day or 300 mg twice a week, or perhaps less, may be sufficient to maintain adequate xanthine oxidase inhibition to reduce serum urate levels. Such patients should be treated with the lowest effective dose, in order to minimize side effects.

Mild reticulocytosis has appeared in some patients.

Periodic determination of liver and kidney function and complete blood counts should be performed especially during the first few months of therapy.

ADVERSE REACTIONS:

Dermatologic: Because in some instances skin rash has been followed by severe hypersensitivity reactions, it is recommended that therapy be discontinued at the first sign of rash or other adverse reaction (see WARNINGS). Skin rash, usually maculopapular, is the adverse reaction most commonly reported. The incidence of skin rash may be increased in the presence of renal disorders.

Exfoliative, urticarial and purpuric lesions, Stevens-Johnson syndrome (erythema multiforme) and toxic epidermal necrolysis have also been reported.

A few cases of alopecia with and without accompanying dermatitis have been reported.

In some patients with a rash, restarting Zyloprim (allopurinol) therapy at lower doses has been accomplished without untoward incident.

Gastrointestinal: Nausea, vomiting, diarrhea, and intermittent abdominal pain have been reported.

Hepatic: Rare cases of granulomatous hepatitis and hepatic necrosis have been reported.

Vascular: There have been rare instances of a generalized hypersensitivity vasculitis or necrotizing angiitis which have led to irreversible hepatotoxicity and death.

Hematopoietic: Agranulocytosis, anemia, aplastic anemia, bone marrow depression, leukopenia, pancytopenia and thrombocytopenia have been reported in patients, most of whom received concomitant drugs with potential for causing these reactions. Zyloprim has been neither implicated nor excluded as a cause of these reactions.

Renal: Rare cases of renal failure have been reported in hypertensive patients who received thiazides and Zyloprim concurrently. Some patients had evidence of hypersensitivity to allopurinol.

Neurologic: There have been a few reports of peripheral neuritis occurring while patients were taking Zyloprim. Drowsiness has also been reported in a few patients.

Ophthalmic: There have been a few reports of cataracts found in patients receiving Zyloprim. It is not known if the cataracts predated the Zyloprim therapy. "Toxic" cataracts were reported in one patient who also received an anti-inflammatory agent; again, the time of onset is unknown. In a group of patients followed by Gutman and Yü for up to five years on Zyloprim therapy, no evidence of ophthalmologic effect attributable to Zyloprim was reported.

Drug Idiosyncrasy: Symptoms suggestive of drug idiosyncrasy have been reported in a few patients. This was characterized by fever, chills, leukopenia or leukocytosis, eosinophilia, arthralgias, skin rash, pruritus, nausea and vomiting.

OVERDOSAGE: Massive overdosing, or acute poisoning, by Zyloprim has not been reported.

HOW SUPPLIED: 100 mg (white) scored tablets, bottles of 100 and 1000; 300 mg (peach) scored tablets, bottles of 30, 100 and 500. Unit dose packs for each strength also available.

Complete information available from your local B. W. Co. Representative or from Professional Services Department PML.

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Pulmonary Carcinoma

(Continued from page 254)

the patient is a good risk surgical candidate — tissue can be obtained at thoracotomy.

Bronchoscopy should be done in all patients prior to thoracotomy.

Low risk individuals presenting with small peripheral lesions need only gross bronchoscopic evaluation prior to thoracotomy, but fine needle aspiration biopsy could be done to help eliminate the need for intraoperative biopsy prior to formal resection.

Bone scans, liver scans, and brain scans are unnecessary in asymptomatic individuals. Positive bone scans and positive liver scans did not correlate well with mild elevations (≤25% of normal values) of serum alkaline phosphatase or liver enzymes.

Chest CT scans should not be used to determine non-resectability but when negative for mediastinal adenopathy, may contradict the need for mediastinal exploration prior to thoracotomy.[15]

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TEFRA

Health Care Coverage for Employees Age 65-69 Years

The Tax Equity and Fiscal Responsibility Act (TEFRA) regulations have yet to be finalized, but there is a potential for significant impact on businesses and their employees.

TEFRA includes amendments to the Social Security Act and the Age Discrimination Employment Act, as well as changes in Medicare.

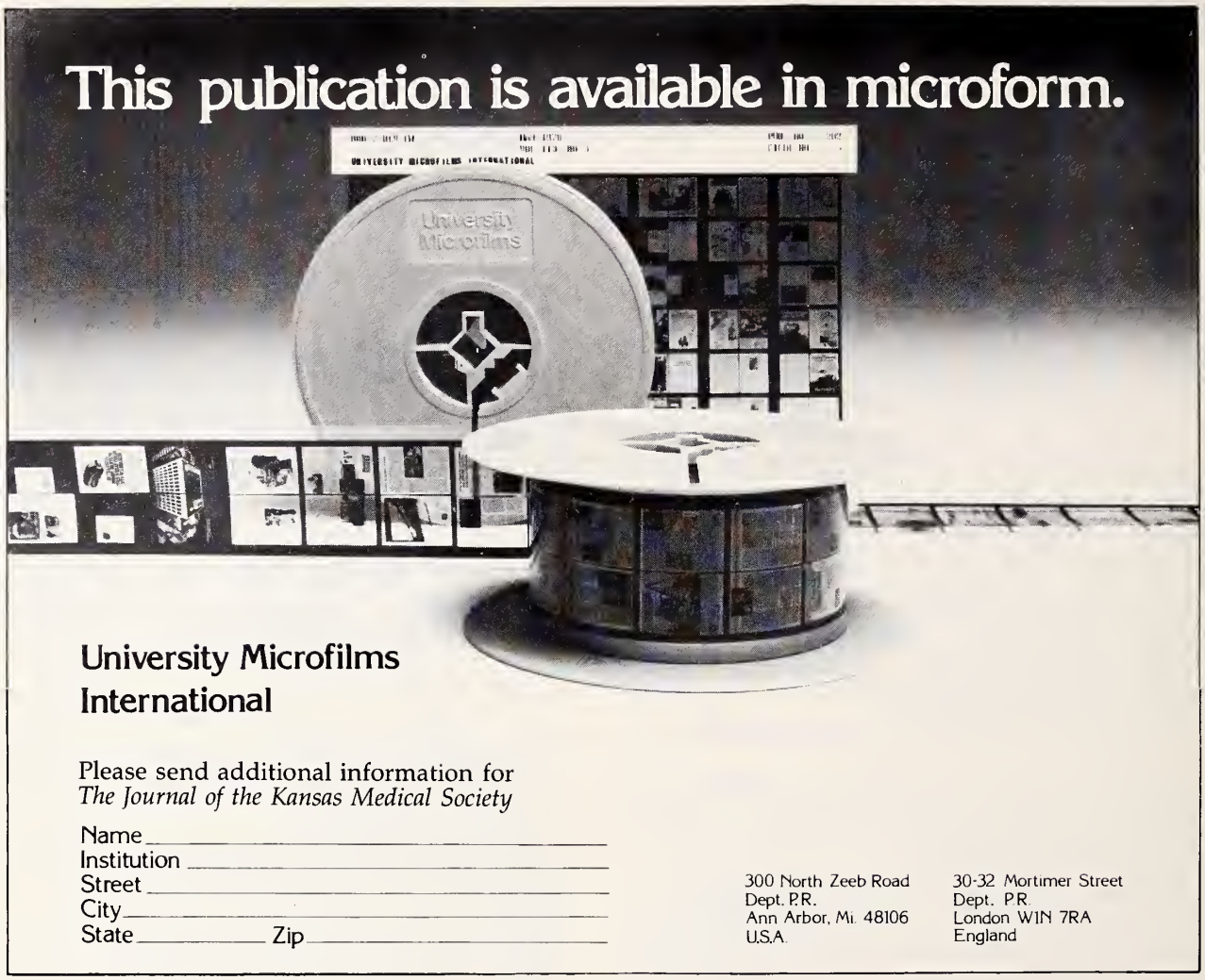
Blue Cross and Blue Shield has advised that the regulations will apply to any employer with 20 or more employees with a group health care plan where there is a company contribution; only employees age 65-69 years will be affected. Employers will probably be allowed 60 days for compliance following publication of the regulations.

The regulations as proposed are as follows:

- Medicare will not pay primary benefits even though the employer's plan states that its benefits are secondary to Medicare.
- Employers must offer to employees age 65-69 years the same health care coverage offered to employees under age 65 years and at the same cost as for younger employees.
- Each eligible employee must be given the opportunity to elect the employer's group health plan as primary coverage *or* to totally reject the employer's plan in favor of Medicare as primary coverage.
- Employees will not be permitted to keep a Medicare Exclusion Rider or Plan 65 coverage in the group. In order to remain in the employer's group health coverage program, the active employee age 65-69 years will need to carry the same coverage as employees under age 65 years. If the employee wants Medicare to be primary, the employer will be required to expend the same amount of money for health care services for that employee as for younger employees.

The spouse of an employee age 65-69 years must be provided with the same coverage that is provided to the spouses of younger employees. This means that a Medicare Exclusion Rider program will still be available to spouses.

An affected employer may wish to consult with an attorney about the impact of these regulations. Until the final rules are adopted, there is no action required; however, employers should be aware of the situation. When the new regulations have been approved and adopted, more specific instructions for bringing coverage into compliance will be made available.



Radiation Therapy

(Continued from page 248)

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for (lorazepam) ℞

Anxiety

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NEW TABLET DESIGN 0.5, 1.0 and 2.0 mg

Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® (IV) for (lorazepam) Anxiety

DOSAGE: Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; Insomnia due to anxiety or transient situational stress, 2-4mg h.s.

HOW SUPPLIED: 0.5, 1.0 and 2.0mg tablets.

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Pediatric Mechanical Ventilators

(Continued from page 262)

purchases to one or two models, so that hospital personnel will be familiar with their peculiarities.

- Ventilators should be periodically checked with a calibrated lung simulator to verify consistency of their performances.
- Safe pediatric ventilator management requires frequent bedside diagnostic evaluation. Serial blood gas values are important data to verify whether machine settings are appropriate, and to assure that optimal gas flow patterns are being provided for the patient with marked ventilatory impedance.

Acknowledgement

Rick Davis provided technical assistance in the preparation of this manuscript.

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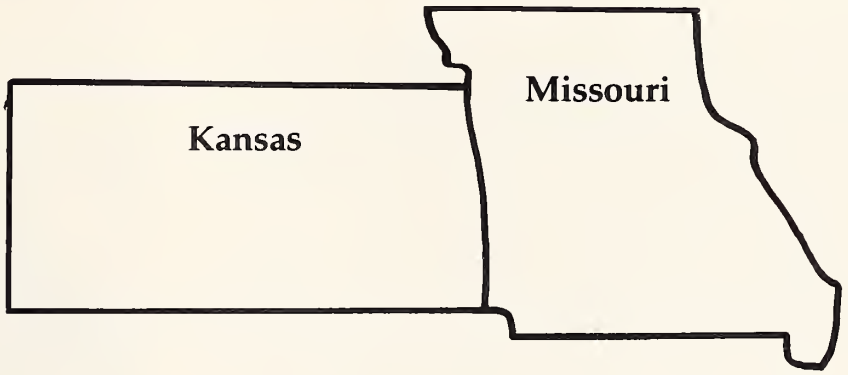
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Penile Implants

(Continued from page 246)

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Bezoars

(Continued from page 250)

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BRIEF SUMMARY
PROCARDIA® (nifedipine) **CAPSULES** For Oral Use

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents: (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients; transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PRDCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PRDCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PRDCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

More detailed professional information available on request.

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Pfizer LABORATORIES DIVISION
PFIZER INC

"I can do things that I couldn't do for 3 yrs. including joining the human race again."



Quotes from an unsolicited letter received by Pfizer from an angina patient. While this patient's experience is representative of many unsolicited comments received, not all patients will respond to Procardia nor will they all respond to the same degree.

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"My daily routine consisted of sitting in my chair trying to stay alive."

"My doctor switched me to PROCARDIA[*] *as soon as it became available. The change in my condition is remarkable."*

"I shop, cook and can plant flowers again."

"I have been able to do volunteer work...and feel needed and useful once again."

PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks,[1] taking fewer nitroglycerin tablets,[2] doing more, and being more productive once again.

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%).



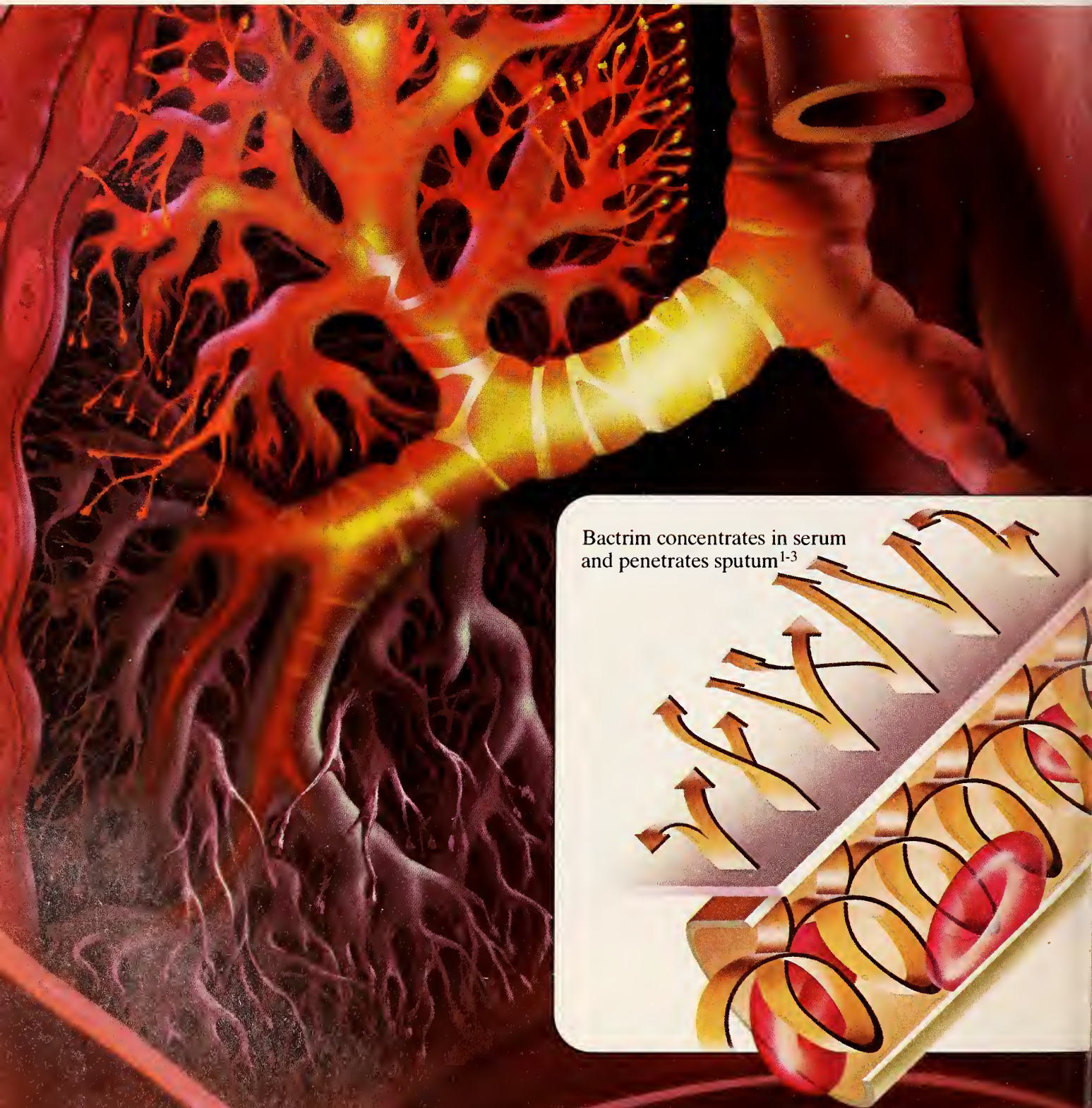
for the varied faces of angina

PROCARDIA®
(NIFEDIPINE) *Capsules 10 mg*

*Procardia is indicated for the management of:
1) Confirmed vasospastic angina.
2) Angina where the clinical presentation suggests a possible vasospastic component.
3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

Please see PROCARDIA *brief summary on adjoining page*

Bactrim™ attacks the
(trimethoprim and sulfamethoxazole/Roche)
in acute exacerbations



Bactrim concentrates in serum and penetrates sputum[1-3]

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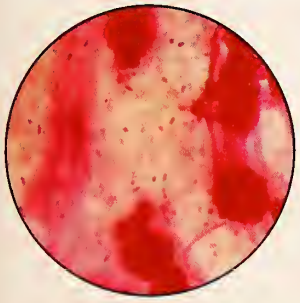
major pathogens of chronic bronchitis*

Bactrim clears sputum of susceptible bacteria

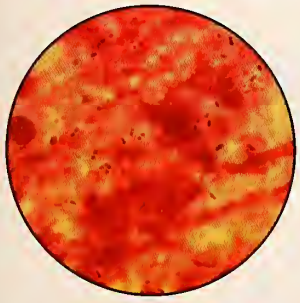
In sputum cultures from patients with acute exacerbations of chronic bronchitis, *H. influenzae* and *S. pneumoniae* are isolated more often than any other pathogens.[4,5] One study of transtracheal aspirates from 76 patients with acute exacerbations found that 80% of the isolates were of these two pathogens.[5]

Bactrim is effective *in vitro* against most strains of both *S. pneumoniae* and *H. influenzae*—even ampicillin-resistant strains. And in acute exacerbations of chronic bronchitis involving these two pathogens, sputum cultures taken seven days after a two-week course of therapy showed that Bactrim eradicated these bacteria in 91% (50 of 55) of the patients treated.[6]

attacks *H. influenzae*—even ampicillin-resistant strains



attacks *S. pneumoniae*



Bactrim reduces coughing and sputum production

In three double-blind comparisons with ampicillin *q.i.d.*, Bactrim DS proved equally effective on all clinical parameters.[7-9] Bactrim reduced the frequency and severity of coughing, reduced the amount of sputum produced and cleared the sputum of purulence.

Bactrim has the added advantages of *b.i.d.* dosage convenience and a lower incidence of diarrhea than with ampicillin, and it is useful in patients allergic to penicillins.

Bactrim also proved more effective than tetracyclines in 10 clinical trials involving nearly 700 patients.[10] Overall clinical condition of the patients, changes in sputum purulence, reduction in sputum volume and microbiological clearance of pathogens—all improved more with Bactrim therapy than with tetracyclines. G.I. side effects occurred in only 7% of patients treated with Bactrim compared with 12% of tetracycline-treated patients. (See Adverse Reactions in summary of product information on next page.)

Bactrim is contraindicated in pregnancy at term and nursing mothers, infants under two months of age, documented megaloblastic anemia due to folate deficiency and hypersensitivity.

Bactrim DS. For acute exacerbations of chronic bronchitis in adults* when it offers an advantage over single-agent antibacterials.

References: 1. Hughes DTD, Bye A, Hodder P: *Adv Antimicrob Antineoplastic Chemother 1*/2:1105-1106, 1971. **2.** Jordan GW *et al: Can Med Assoc J 112*:91S-95S, Jun 14, 1975. **3.** Beck H, Pechere JC: *Prog Antimicrob Anticancer Chemother 1*:663-667, 1969. **4.** Quintiliani R: Microbiological and therapeutic considerations in exacerbations of chronic bronchitis, in *Chronic Bronchitis and Its Acute Exacerbations: Current Diagnostic and Therapeutic Concepts;* Princeton Junction, NJ, Communications Media for Education, Inc., 1980, pp. 9-12. **5.** Schreiner A *et al: Infection 6*(2):54-56, 1978. **6.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **7.** Chodosh S: Treatment of acute exacerbations of chronic bronchitis: results of a double-blind crossover clinical trial, in *Chronic Bronchitis and Its Acute Exacerbations: Current Diagnostic and Therapeutic Concepts. Op. cit.*, pp. 15-16. **8.** Chervinsky P: Double-blind clinical comparisons between trimethoprim-sulfamethoxazole (Bactrim™) and ampicillin in the treatment of bronchitic exacerbations. *Ibid.*, pp. 17-18. **9.** Dulfano MJ: Trimethoprim-sulfamethoxazole vs. ampicillin in the treatment of exacerbations of chronic bronchitis. *Ibid.*, pp. 19-20. **10.** Medici TC: Trimethoprim-sulfamethoxazole (Bactrim™) in treating acute exacerbations of chronic bronchitis: summary of European clinical experience. *Ibid.*, pp. 13-14.

Economical b.i.d.

Bactrim™ DS

(160 mg trimethoprim and 800 mg sulfamethoxazole/Roche)

*Due to susceptible organisms. **Please see next page for summary of product information.**

Bactrim™

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:

Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS:

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100, Prescription Paks of 20 and 28. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. *Pediatric Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz (1 pint). *Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).

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Join

KaMPAC

Sickle Cell Anemia

(Continued from page 270)

Answers

1. *False*
2. *True*
3. *True*
4. *True*
5. *False*

Suggested Readings

1. Davis, J. R.; Vichinsky, E. P. and Lubin, B. H.: Current treatment of sickle cell disease. *Curr. Probl. Pediatr.* 10:1-64, 1980.

2. Dean, J. and Schechter, A. N.: Sickle cell anemia: Molecular and cellular bases of therapeutic approaches. *N. Engl. J. Med.* 299:752-763, 804-811, 863-870, 1978.

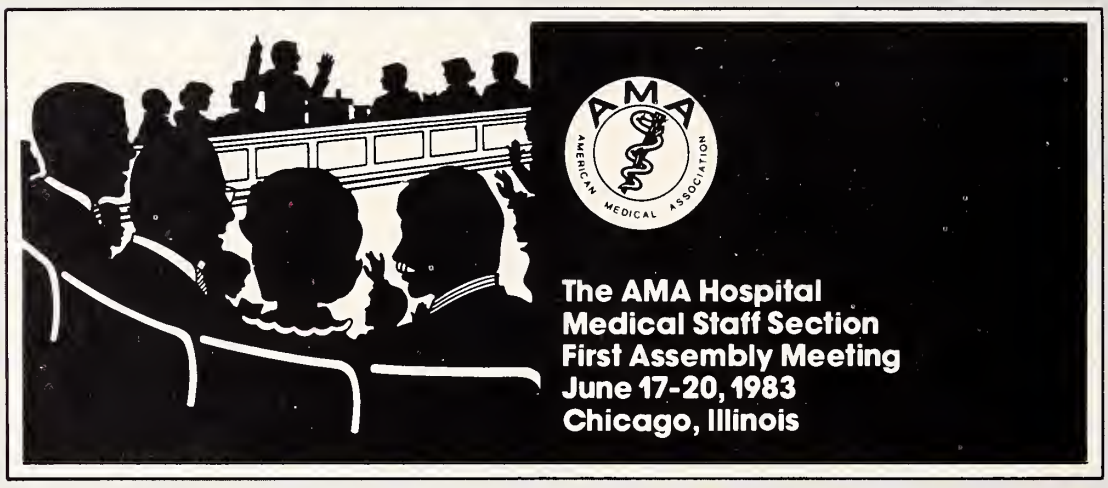
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4. Foster, H. W.: Contraceptives in sickle cell disease. *South. Med. J.* 74:543-545, 1981.

5. Konetey-Ahulu, F. I. D.: The sickle cell diseases. *Arch. Intern. Med.* 133:611-619, 1974. (One of 17 articles re: sickle cell disease. *Arch. Intern. Med.* 133:529-706, 1974.)

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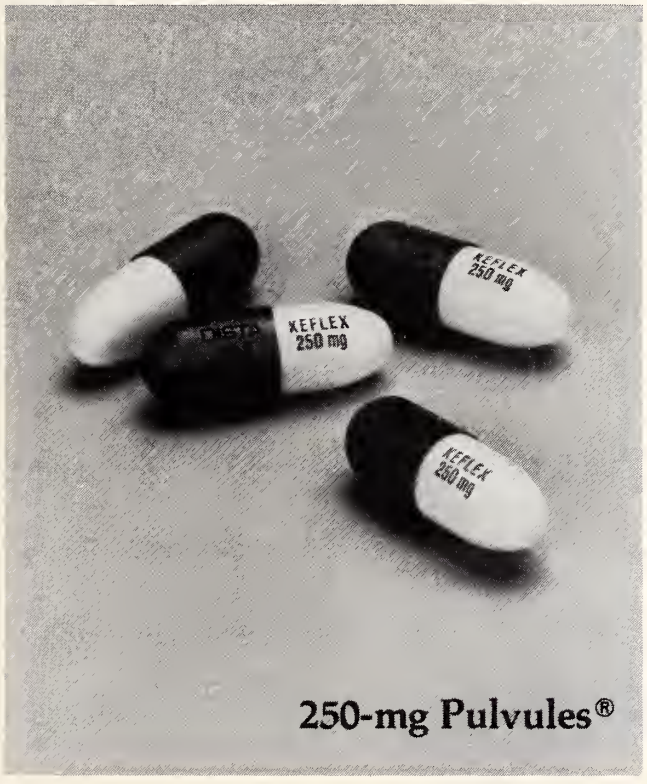
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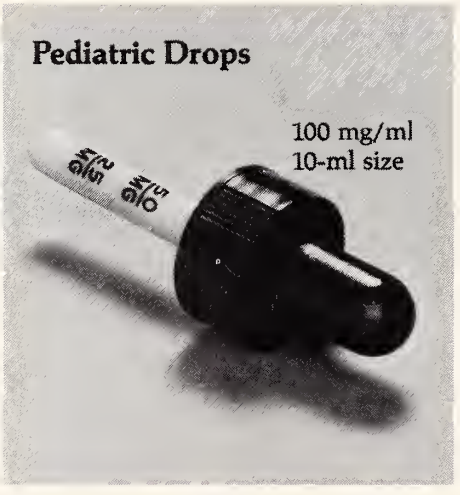
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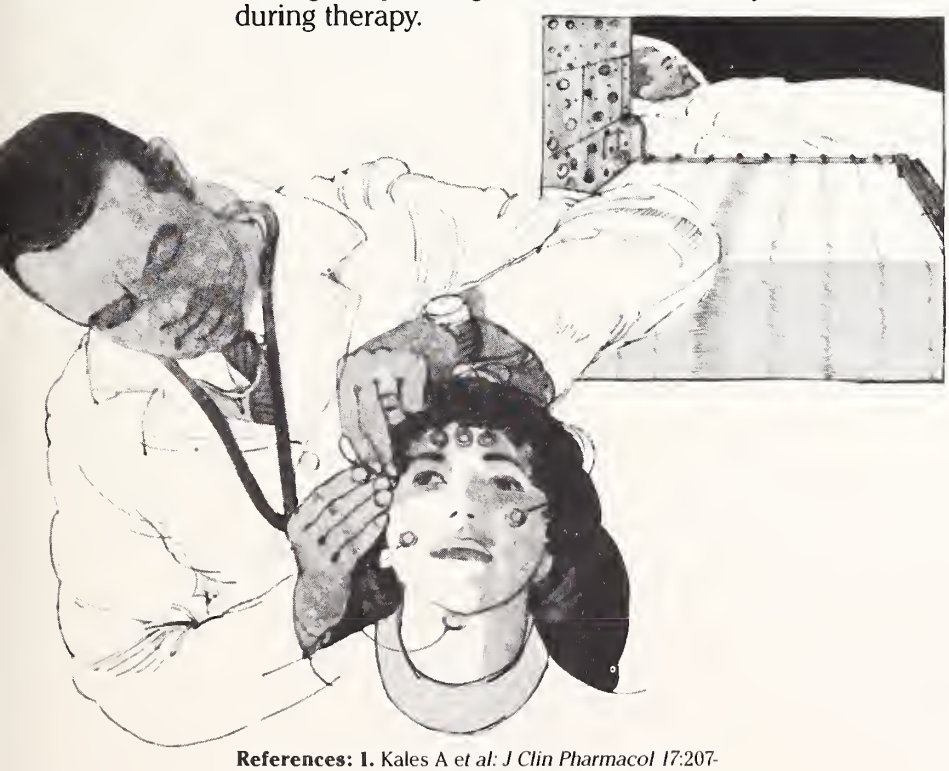
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- Caution patients about drinking alcohol, driving or operating hazardous machinery during therapy.



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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended. Repeated therapy should only be undertaken with appropriate patient evaluation.

Contraindications: Known hypersensitivity to flurazepam HCl; pregnancy. Benzodiazepines may cause fetal damage when administered during pregnancy. Several studies suggest an increased risk of congenital malformations associated with benzodiazepine use during the first trimester. Warn patients of the potential risks to the fetus should the possibility of becoming pregnant exist while receiving flurazepam. Instruct patient to discontinue drug prior to becoming pregnant. Consider the possibility of pregnancy prior to instituting therapy.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg recommended initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

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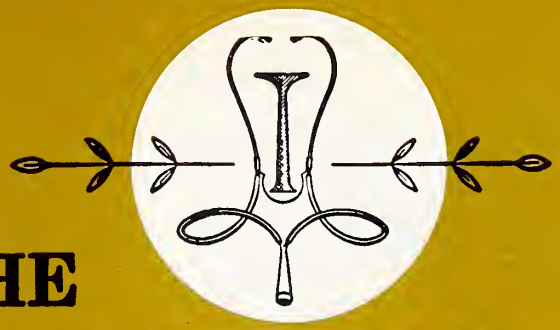
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Commendation for the Douglas County Medical Society & Auxiliary

RESOLUTION NO. 83-17

WHEREAS, The Douglas County Medical Society and its Auxiliary have done an excellent job in hosting the House of Delegates of the Kansas Medical Society and the Auxiliary to the Kansas Medical Society; and

WHEREAS, They have provided excellent facilities for the meeting and seen to the comfort of the members and guests; and

WHEREAS, The athletic, scientific, and social desires of this Society and its Auxiliary have been fulfilled in grand fashion; therefore be it

Resolved, That the House of Delegates here assembled commend the Douglas County Medical Society and its Auxiliary for their splendid effort by a standing vote of applause; and be it further

Resolved, That copies of this resolution be forwarded to the Douglas County Medical Society and its Auxiliary.

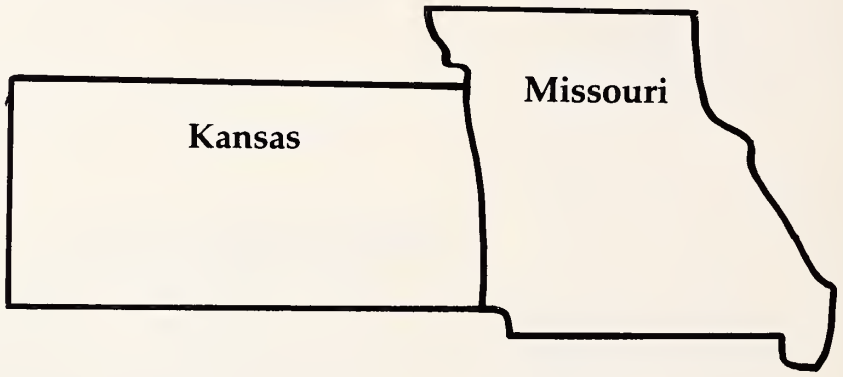
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The Kansas Medical Society has a membership of 2,884 and the Kansas Medical Auxiliary has a membership of 1,274. The following physicians and spouses have elected to become members of KaMPAC and to support "organized medicine" in its efforts to elect "friends of medicine." This listing will be expanded each month to include new KaMPAC members.

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References:

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Relief of mild to moderate pain. Treatment of primary dysmenorrhea.

CONTRAINDICATIONS: Patients hypersensitive to ibuprofen, or with the syndrome of nasal polyps, angio-edema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory drugs (see WARNINGS).

WARNINGS: Anaphylactoid reactions have occurred in patients hypersensitive to aspirin (see CONTRAINDICATIONS). Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Peptic ulceration, perforation, or gastrointestinal bleeding can end fatally, however, an association has not been established. Rufen should be given under close supervision to patients with a history of upper gastrointestinal tract disease, and only after consulting the ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be attempted. If Rufen must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

PRECAUTIONS: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If developed, discontinue Rufen and administer an ophthalmologic examination.

Fluid retention and edema have been associated with Rufen; caution should be used in patients with a history of cardiac decompensation.

Rufen can inhibit platelet aggregation and prolong bleeding time. Use with caution in patients with intrinsic coagulation defects and those taking anticoagulants.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy, this therapy should be tapered slowly when adding Rufen.

DRUG INTERACTION: *Coumarin-type anticoagulants.* The physician should be cautious when administering Rufen to patients on anticoagulants.

Aspirin. Concomitant use may decrease Rufen blood levels.

PREGNANCY AND NURSING MOTHERS: Rufen should not be taken during pregnancy nor by nursing mothers.

ADVERSE REACTIONS: Incidence greater than 1%. Gastrointestinal: The most frequent adverse reaction is gastrointestinal (4 to 16%). Includes nausea*, epigastric pain*, heartburn*, diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence). Central Nervous System: dizziness*, headache, nervousness. Dermatologic: rash* (including maculopapular type), pruritus. Special Senses: tinnitus. Metabolic: decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS). *Incidence 3% to 9%.

Incidence less than 1 in 100. Gastrointestinal: gastric or duodenal ulcer with bleeding and/or perforation, hemorrhage, melena. Central Nervous System: depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma. Dermatologic: vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome and alopecia. *Special Senses:* hearing loss, amblyopia (blurred and/or diminished vision, scotomata and/or changes in color vision) [see PRECAUTIONS]. Hematologic: neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs' positive), thrombocytopenia with or without purpura eosinophilia, decreases in hemoglobin and hematocrit. Cardiovascular: congestive heart failure in patients with marginal cardiac function, elevated blood pressure. Allergic: syndrome of abdominal pain, fever, chills, nausea and vomiting, anaphylaxis, bronchospasms (see CONTRAINDICATIONS). Renal: acute renal failure in patients with preexisting significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria. Miscellaneous: dry eyes and mouth, gingival ulcers, rhinitis.

Causal relationship unknown. Gastrointestinal: pancreatitis. Central Nervous System: paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri. Dermatologic: toxic epidermal necrolysis, photoallergic skin reactions. Special Senses: conjunctivitis, diplopia, optic neuritis. Hematologic: bleeding episodes. Allergic: serum sickness, lupus erythematosus syndrome, Henoch-Schonlein vasculitis. Endocrine: gynecomastia, hypoglycemia. Cardiovascular: arrhythmias (sinus tachycardia, bradycardia, and palpitations). Renal: renal papillary necrosis.

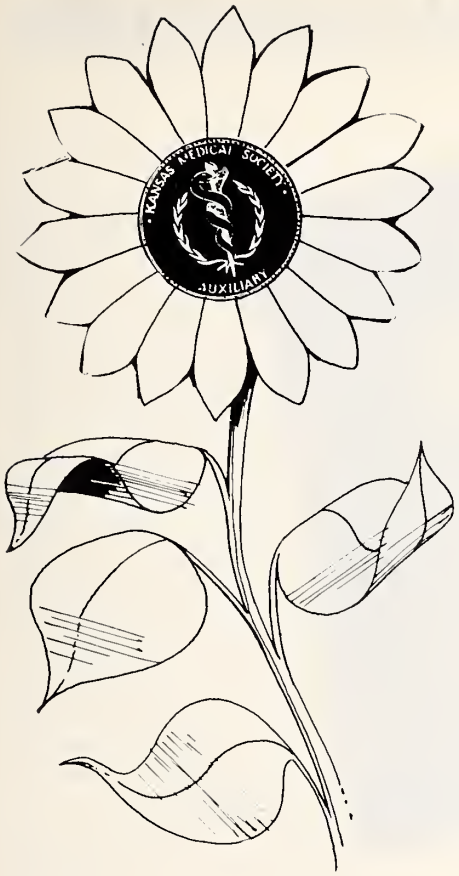
OVERDOSAGE: Acute overdosage, the stomach should be emptied. Rufen is acidic and excreted in the urine, alkaline diuresis may benefit.

DOSAGE AND ADMINISTRATION: Rheumatoid arthritis and osteoarthritis, including flareups of chronic disease: Suggested dosage 400 mg t.i.d. or q.i.d.

Dysmenorrhea: 400 mg every 4 hours as necessary.

Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for the relief of pain. *Do not exceed 2,400 mg per day.*

CAUTION: Federal law prohibits dispensing without prescription.



AUXILIARY NEWS

An Open Letter to Kansas Physicians

The Kansas Medical Society Auxiliary Convention in Lawrence, May 5-8, was a terrific success. Shirley Wilcox (Howard), publicity chairman for the convention, will chronicle the Auxiliary's activities in next month's convention issue of the *Journal*. Still, I must now thank Markie Bieri (Peter), Convention Chairman; Barbara Learned (George), Chairperson and Founder of the Sports Day for the Non-Athlete and the Athlete; Kitty Gray (Captain), County President; and all 60-plus Douglas County Auxiliary members. I have never witnessed a group work so well together and have such a grand time as well. I am extremely proud and pleased to be a member of this Auxiliary.

Steve Carter, Executive Director of the Kansas Medical Society, presented a proposal to the post-convention board for a project on aging. The joint venture of the Society and the Auxiliary would focus on staging county forums on the subject of the economic mechanics of aging. The forums would deal with such subjects as reimbursement and coverage of various medical problems by Medicare and the correct procedure in filling out Medicare forms. The program will be organized and structured by the Kansas Medical Society office and the Auxiliaries will handle the details at the county level. Hopefully, with this program the public will view the medical community as facilitators rather than reactors to government programs.

Details of the project will be outlined at the Auxiliary Summer Board Meeting in Lawrence, July 13-14. We need participation from the entire membership and welcome all. The Medical Society is organizing an informative seminar on aging scheduled for the afternoon of July 13, and the workshops and board meeting will be on July 14. Please invite your spouse to attend. Shirley Reese (John), 2417 Princeton, Lawrence KS 66044, is the meeting chairperson; please contact her for details.

The National Auxiliary Convention, in conjunction with the AMA Convention, will be June 19-22 at the Drake Hotel, Chicago. Our delegates are Meldon Laury (David), Ann Rempel (John), Caryl Bichlmeier (Frank), Betty Glover (Richard), and myself. Alternates are Dot Meyer (Warren), Kathy Wedel (Kermit), Nancy Macy (Ted), and Betty Moore (Robert).

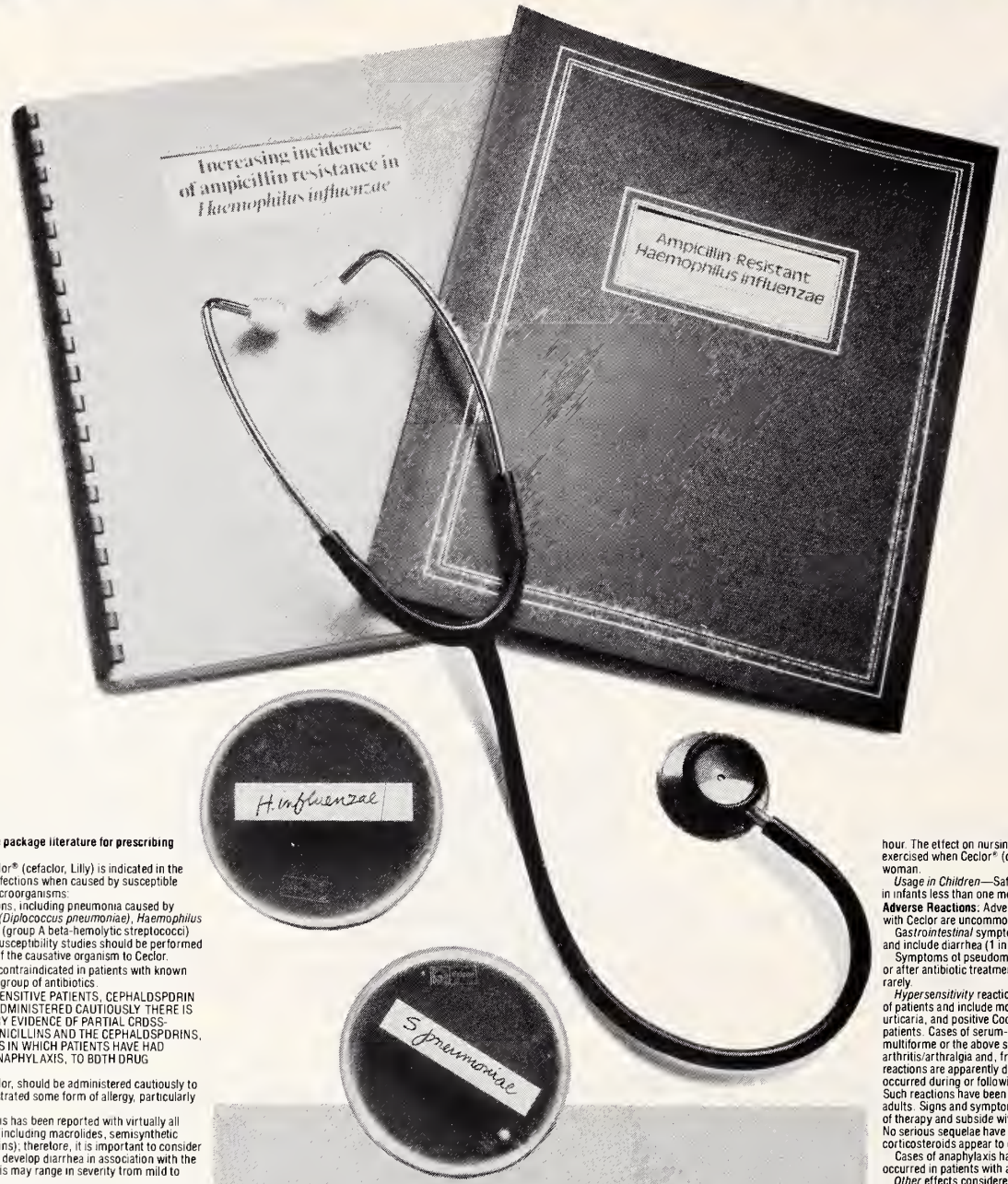
On Sunday, June 19, there will be workshops on AMAERF, Health Projects, Legislation, and Membership. Each participant can attend two workshops. Ambassador John G. Volpe (Ret.), Chairman of the Presidential Commission on Drunk Driving, will be a featured speaker as will author Shana Alexander. For Kansans, the highlight speaker will be Senator Nancy Kassebaum.

Two of our members have positions at the national level: Jean Cavanaugh (Clair) is a North Central Regional Director, and Lela May Young (Chester) is an active Past National President. We are very fortunate to have such excellent leadership from Kansas.

Attending a national convention is a very inspirational and enriching experience. We gain the tools to enable us to lead and coordinate programs at the state and local levels.

Diane Sanders
President
Kansas Medical Society Auxiliary

An added complication... in the treatment of bacterial bronchitis*



Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-5]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

Ceclor®
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Pulvules®, 250 and 500 mg

Brief Summary. Consult the package literature for prescribing information.

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae,* and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile.* Other causes of colitis should be ruled out.

Precautions: *General Precautions*—If an allergic reaction to Ceclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip; USP; Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers—Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor® (cefaclor, Lilly) is administered to a nursing woman.

Usage in Children—Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below:

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations of SGDT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

[061782R]

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae.*[8]
Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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7. Data on file, Eli Lilly and Company.
8. Principles and Practice of Infectious Diseases (edited by G.L. Mandell, R.G. Douglas, Jr., and J.E. Bennett), p. 487. New York: John Wiley & Sons, 1979.

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Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

300035

Herpes Simplex Virus

Diagnosis and Management in Pregnant Women

Recent concerns of medical and lay populations concerning the risk of transmission of herpes simplex virus (HSV), type I and II, to the fetus and neonate of a mother with active genital and cutaneous lesions have stimulated development of valid methods of diagnosis and treatment. The May/June issue of the *Iowa Perinatal Letter* summarizes risks, diagnosis, and treatment of "Genital Herpes in the Pregnant Woman" and "Neonatal Herpes Simplex Virus Infection." The Medical Council of the Kansas Perinatal Care Program and the Bureau of Maternal and Child Health, Kansas Department of Health and Environment, consider the recommendations of these articles worthy of review by Kansas physicians. With permission from the *Iowa Perinatal Letter,* the material has been revised to reflect Kansas circumstances.

Genital Herpes in the Pregnant Woman

Michael W. Varner, M.D.

Genital herpes is one of the most common venereal diseases in the United States. In fact, several published reports suggest that it is now more common than gonorrhea in private practice settings. In pregnancy there exists a definite risk of vertical transmission from mother to fetus. Risks of genital herpes in the pregnant woman and currently available methods of diagnosis and management are reviewed here.

Risks

Because there is no national surveillance system, the absolute incidence of maternal genital herpes infection remains unknown. It has been estimated that, in certain populations, as many as one in every 150 pregnant women sheds herpes simplex virus (HSV) during pregnancy. No specific incidence figures are available for patients in Kansas, but the risk of maternal genital herpes here is probably less than 1/150 pregnancies. Many patients are asymptomatic, and some have no previous history of symptoms.

Retrospective studies have shown that newborns who develop HSV infections, presumably from colonized or infected maternal tissues during parturition, stand approximately a 50 per cent risk of dying in the neonatal period. Among those who survive, the risk of subsequent neurologic impairment is high. Although only 1/5,000 to 1/20,000 newborns acquires neonatal herpes, this still represents two to eight infants per year in Kansas and roughly 200-800 infants per year nationally.

Studies have shown that abdominal delivery can diminish, but not eliminate, the risk of neonatal herpes. Although much less common, apparent herpes simplex infections during the first trimester have been associated with fetal structural anomalies.

Diagnosis

Any pregnant woman with a history of painful vesicular genital lesions should be suspected of having genital herpes. These lesions have an edematous, erythematous base and are frequently accompanied by symptoms of constitutional illness as well as local lymphadenopathy. Neurologic dissemination is rare but can occur. The vesicles frequently open and become ulcerated. They usually persist for seven to ten days and are contagious until they re-epithelialize. Although intravaginal or cervical herpes lesions are rare, 30-50 per cent of all symptomatic patients will have positive cervical cultures.

Recurrent genital herpes infections generally have a shorter duration of clinical illness, usually lasting from one to ten days.

In either primary or recurrent infections, diagnosis is best accomplished using tissue culture techniques. Swab specimens should be obtained from the base of suspicious fresh lesions as well as from the cervix. Several Kansas virology laboratories, including the Office of Laboratories and Research, Kansas Department of Health and Environment, Forbes Field, Topeka, KS 66620 (913-862-9360), employ cell culture systems for the isolation of HSV. Specimens can be mailed to the state laboratory using transport media. The virus will remain stable in a buffered viral transport medium which may be purchased through commercial laboratory supply companies. If a commercial medium is not immediately available, the state virology laboratory will supply it on a temporary basis. The formula used to prepare this medium can be found in the *Health Laboratory Services Manual* published by the State Office of Laboratories. Inoculated transport medium should be shipped either with ice packs or frozen with dry ice at −57C. Culture results should be available 24-72 hours after receipt by the state laboratory. For further information contact Patrick Hays, Ph.D., Offices of Laboratories and Re-

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search at the address or telephone number (Ext. 416) given above.

Several recommendations have been made regarding cervical culture protocols in pregnant women with a history of genital herpes. The current policy involves weekly cultures of the cervix and vulva as well as any suspicious lesions during the last month of pregnancy. However, if the pregnant woman with a history of recurrent herpes has no evidence of active lesions and two documented consecutive negative cultures during the final month of pregnancy, additional cultures are not suggested. Isolates from two separate sites can be placed in one viral transport tube, reducing culture costs.

The diagnostic virus laboratory should be used to confirm a diagnosis of suspected herpes simplex infection in a pregnant woman. However, it is also appropriate that the limitations of culturing be reviewed. A negative laboratory report does not guarantee that the virus was absent at the time the specimen was obtained. Improper collection, storage, or transportation of the specimen to the laboratory will occasionally give a false negative report since this virus is labile even under ideal conditions. Infection may also occur after the last specimen has been collected.

Indirect methods such as Pap smear preparations that identify multinucleated giant cells may provide indirect evidence of HSV infection or colonization in 60-80 per cent of cases. These techniques are neither as sensitive nor as specific as the preferred culture techniques.

Antibody tests are available, but are mentioned only to emphasize their limitation to primary infections, with seroconversions being diagnostic, while being useless in following patients with histories of genital herpes infection. The majority of the population have demonstrable antibodies, and the presence of antibodies neither protects against recurrent maternal herpes nor confers total protection against neonatal herpes as is the case with rubella and toxoplasmosis maternal antibodies.

When a first trimester infection is encountered and systemic symptoms suggest that viremia may

Anxious patients improve in just a few days



And what is more reassuring to an excessively anxious patient than medication that promptly starts to relieve his discomforting symptoms? Valium® (diazepam/Roche) begins working within 30 to 90 minutes. Patients continue to improve in just a few days, and relief continues throughout the course of treatment.

There are other important benefits with Valium as well—along with its broad clinical range, Valium has an efficacy/safety profile that few, if any, drugs can match. This record has been achieved with extensive clinical experience, undoubtedly including yours. And, as you must have observed, side effects more serious than drowsiness, fatigue or ataxia rarely occur. Nevertheless, as with any CNS-acting agent, patients should be cautioned about driving, operating hazardous machinery or ingesting alcohol or other CNS-depressant drugs while taking Valium.

Yet another benefit Valium affords is flexibility. Available in 2-mg, 5-mg and 10-mg scored tablets, Valium enables you to titrate dosage to individual patient needs. For the geriatric patient, a starting dosage of 2 to 2½ mg once or twice a day is recommended. And, for patients who forget or skip medication, you can prescribe Valrelease™ (diazepam/Roche) 15-mg slow-release capsules, knowing that Valrelease will assure all the benefits of Valium 5 mg *t.i.d.* with the convenience of once-a-day dosage.

Discontinuation of Valium (or Valrelease) is typically as smooth as its start in short-term therapy. However, Valium and Valrelease should be discontinued gradually after more extended treatment. As you diminish dosage, the built-in tapering action of Valium and Valrelease will help avoid rapidly recurring anxiety symptoms and symptoms of withdrawal, and will help ease the patient's transition to independent coping when therapeutic goals have been achieved.

...that's one of the unique benefits of

Valium®

diazepam/Roche

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For a summary of product information, please turn the page.

ROCHE®

Valium® (diazepam/Roche) Ⓝ Tablets
Valrelease™ (diazepam/Roche) Ⓝ slow-release Capsules
Injectable Valium® (diazepam/Roche) Ⓝ

Before prescribing, **please consult complete product information, a summary** of which follows:

Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety. Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic. Symptomatic relief of acute agitation, tremor, impending or acute delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in: relief of skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome. *Oral forms* may be used adjunctively in convulsive disorders, but not as sole therapy. *Injectable form* may also be used adjunctively in: status epilepticus; severe recurrent seizures; tetanus; anxiety, tension or acute stress reactions prior to endoscopic/surgical procedures; cardioversion.

The effectiveness of diazepam in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindications: Tablets or capsules in children under 6 months of age; known hypersensitivity; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence.

> *Usage in Pregnancy:* **Use of minor tranquilizers during first trimester should almost always be avoided because their use is rarely a matter of urgency and because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

ORAL: Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral forms adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures.

INJECTABLE: *To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling and, rarely, vascular impairment when used I.V.: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veins,* i.e., *dorsum of hand or wrist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Injectable Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.*

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea; have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs.

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status. Not recommended for OB use.

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of diazepam, *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed and tolerated).

The clearance of diazepam and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

INJECTABLE: Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated.

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug.

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, observed in patients during and after diazepam therapy are of no known significance.

INJECTABLE: Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia.

In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported.

Dosage: Individualize for maximum beneficial effect.

ORAL: *Adults:* Anxiety disorders, relief of symptoms of anxiety—Valium (diazepam/Roche) tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 Valrelease capsules (15 to 30 mg) daily. Acute alcohol withdrawal—tablets, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; or 2 capsules (30 mg) the first 24 hours, then 1 capsule (15 mg) daily as needed. Adjunctively in skeletal muscle spasm—tablets, 2 to 10 mg t.i.d. or q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily. Adjunctively in convulsive disorders—tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily.

Geriatric or debilitated patients: Tablets—2 to 2½ mg 1 or 2 times daily initially, increasing as needed and tolerated (see Precautions). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose.

Children: Tablets—1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use in children under 6 months). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose (not for use in children under 6 months).

INJECTABLE: Usual initial dose in older children and adults is 2 to 20 mg I.M. or I.V., depending on indication and severity. Larger doses may be required in some conditions (tetanus). In acute conditions injection may be repeated within 1 hour, although interval of 3 to 4 hours is usually satisfactory. Lower doses (usually 2 to 5 mg) with slow dosage increase for elderly or debilitated patients and when sedative drugs are added. (See Warnings and Adverse Reactions.)

For dosages in infants and children see below; have resuscitative facilities available.

I.M. use: by deep injection into the muscle.

I.V. use: inject slowly, take at least one minute for each 5 mg (1 ml) given. Do not use small veins, i.e., *dorsum of hand or wrist. Use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.*

Moderate anxiety disorders and symptoms of anxiety, 2 to 5 mg I.M. or I.V., and severe anxiety disorders and symptoms of anxiety, 5 to 10 mg I.M. or I.V., repeat in 3 to 4 hours if necessary; acute alcohol withdrawal, 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary. Muscle spasm, in adults, 5 to 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary (tetanus may require larger doses); in children *administer I.V. slowly*; for tetanus in infants over 30 days of age, 1 to 2 mg I.M. or I.V., repeat every 3 to 4 hours if necessary; in children 5 years or older, 5 to 10 mg repeated every 3 to 4 hours as needed. Respiratory assistance should be available.

Status epilepticus, severe recurrent convulsive seizures (I.V. route preferred), 5 to 10 mg adult dose administered slowly, repeat at 10- to 15-minute intervals up to 30 mg maximum. Repeat in 2 to 4 hours if necessary, keeping in mind possibility of residual active metabolites. Use caution in presence of chronic lung disease or unstable cardiovascular status. Infants (over 30 days) and children (under 5 years), 0.2 to 0.5 mg slowly every 2 to 5 min., up to 5 mg (I.V. preferred). Children 5 years plus, 1 mg every 2 to 5 min., up to 10 mg (slow I.V. preferred); repeat in 2 to 4 hours if needed. EEG monitoring may be helpful.

In endoscopic procedures, titrate I.V. dosage to desired sedative response, generally 10 mg or less but up to 20 mg (if narcotics are omitted) immediately prior to procedure; if I.V. cannot be used, 5 to 10 mg I.M. approximately 30 minutes prior to procedure. As preoperative medication, 10 mg I.M.; in cardioversion, 5 to 15 mg I.V. within 5 to 10 minutes prior to procedure. Once acute symptomatology has been properly controlled with injectable form, patient may be placed on oral form if further treatment is required.

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension. Dialysis is of limited value.

How Supplied:

ORAL: Valium scored tablets—2 mg, white; 5 mg, yellow; 10 mg, blue—bottles of 100 and 500; Prescription Paks of 50, available in trays of 10; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25 and in boxes containing 10 strips of 10.

Valrelease (diazepam/Roche) slow-release capsules—15 mg (yellow and blue), bottles of 100; Prescription Paks of 30.

INJECTABLE: Ampuls, 2 ml, boxes of 10; Vials, 10 ml, boxes of 1; Tel-E-Ject® (disposable syringes), 2 ml, boxes of 10. Each ml contains 5 mg diazepam, compounded with 40% propylene glycol, 10% ethyl alcohol, 5% sodium benzoate and benzoic acid as buffers, and 1.5% benzyl alcohol as preservative.

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have occurred, amniocentesis for viral culture can be considered.

Management

Management of pregnant patients is directed toward preventing contact between the fetus and infected or colonized maternal tissues. Management includes weekly virus isolation testing during the last month of pregnancy and is recommended for asymptomatic patients who have had herpes or who have had contact with a partner who has had herpes. Some authorities also recommend herpes cultures for patients with evidence of other sexually transmitted diseases. The fetus should be delivered abdominally if the culture was positive within the final two weeks and the membranes ruptured less than four hours. If, however, there is no evidence of active lesions, and cultures were negative within the last two weeks, conservative management may be considered. It must be emphasized that abdominal delivery can reduce, but not eliminate, the risk of neonatal herpes. This should be pointed out to the patient and her family.

A pregnant woman with herpes infection involving only the thighs or buttocks should be surveyed in the same manner as described above. These perigenital infections may be associated with unrecognized, asymptomatic genital colonization. If there is no evidence of genital infection for two weeks prior to delivery, such pregnancies can be delivered vaginally, but special precautions must be taken to protect the child from contact with the infected areas.

Recently a new drug, acyclovir (Zovirax), has been approved by the FDA for treatment of herpes infections in immunosuppressed patients only. It has not been shown to decrease length or severity of illness or communicability in otherwise healthy patients. It is not approved for use in pregnancy.

There is also evidence to suggest that adenine arabinoside therapy can decrease mortality rates in infants with neonatal herpes infections. However, this agent is not indicated during pregnancy.

Current therapy for genital herpes in the pregnant

woman remains symptomatic and is directed toward keeping the area clean and dry.

Neonatal Herpes Simplex Virus Infection

Donna E. Bosworth, M.D.

Herpes simplex virus (HSV) causes cold sores, gingivostomatitis, keratitis, encephalitis, and genital ulcers in children and adults. Neonatal HSV infection remains an uncommon (1/10,000 newborns) but devastating illness. Despite the recent availability of antiviral therapy, one of three affected neonates dies and more than one-half of the survivors suffer permanent neurologic or ocular sequelae.

Microbiology and Epidemiology

HSV exists as two strains, type 1 and type 2. Type 1 HSV (oral strain) usually causes mouth lesions, eye infections, or encephalitis and is primarily transmitted by the nonvenereal route; type 2 HSV (genital strain) causes genital infection and is primarily transmitted venereally. However, each type can affect all sites and be transmitted by either route. Type 2 HSV causes 75 per cent of neonatal HSV infections; type 1 HSV is responsible for the remainder. The mother is the most important known source of HSV for the newborn, and the infected maternal genital tract is the primary site of exposure to the virus. In a small percentage of neonatal cases, nongenital sites and exposures to family members, hospital personnel, or other hospitalized infants are sources of HSV. The risk of infection is highest in infants born to culture positive women at delivery, to those with primary as opposed to recurrent genital infection, and to those whose fetal membranes have been ruptured for longer than four hours. If clincially recognized or cytologically or virologically confirmed genital HSV exists at the time of vaginal or delayed cesarean section delivery, the risk of neonatal infection is 50 per cent. In contrast, infants born to an infected mother by cesarean section without rupture of membranes incur a less than 5 per cent risk of neonatal infection.

Pathogenesis

Herpes simplex virus infects the neonate in three ways: ascending infection from the maternal genital tract; contact with the infected genital tract during vaginal delivery; and physical contact with infected persons in the perinatal period. Probable portals of entry include the skin, eye, umbilical cord, and upper respiratory tract. Postnatally, infants acquire virus from individuals with mucocutaneous herpes or from the hands of nursery personnel. Infection can localize at mucocutaneous sites, but in the majority of cases, virus disseminates to viscera or central nervous system (CNS) by the hematogenous and neural routes.

Clinical Manifestations

Greater than 95 per cent of infants with HSV infection manifest signs. The average age of onset is 10-20 days. Neonates present with nonspecific clinical findings resembling bacterial sepsis: fluctuations in temperature, lethargy, jaundice, respiratory distress and cyanosis, poor feeding, vomiting, and CNS signs. In two-thirds of cases, virus disseminates to the viscera with or without CNS involvement. In the remaining one-third of cases, infection localizes to the CNS, skin, eyes, or mouth. Disseminated infection involves the liver (hepatitis), lung (pneumonitis), and adrenals (shock, DIC). In addition, 50 per cent of these infants manifest signs of CNS involvement: irritability, bulging fontanelle, seizures, changes in muscle tone, or coma. A vesicular eruption is the cutaneous hallmark of neonatal HSV infection and exists in 70 per cent of cases sometime during the illness. Typically, two or more grouped 1-2 mm vesicles develop on an erythematous base. Lesions characteristically appear at the cutaneous portal of entry of the virus; for instance, the infant's presenting part (scalp, buttocks) contiguous with the infected maternal genital tract or at areas of skin trauma (scalp electrode, EKG, and temperature monitors). The mucocutaneous manifestations of HSV — vesicular rash, oral ulcers, and keratoconjunctivitis — occur alone, but more commonly appear prior to or in conjunction with progressive disseminated infection.

Diagnosis

HSV infection is readily diagnosed if characteristic skin, oral, or eye lesions are recognized. However, only one-third of patients initially manifest these signs of illness. As many as one-third of affected infants never show cutaneous, oral, or ocular manifestations during their infection. In the absence of these mucocutaneous stigmata, diagnosis is difficult and often delayed. HSV infection should be considered in the neonate with suspected bacterial sepsis whose bacterial cultures are negative or in the infant with signs of encephalitis. In addition to bacterial sepsis, the differential diagnosis of HSV infection includes neonatal enterovirus and cytomegalovirus infections.

Epidemiologic information on the mother and her sexual contacts contributes to the diagnosis of neonatal HSV infection. For example, mothers of

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Agent of change

Ativan®

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Anxiety

See important information on following page.

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NEW TABLET DESIGN 0.5, 1.0 and 2.0 mg

Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® (IV) for (lorazepam) Anxiety

DOSAGE: Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.

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neonates with HSV infection may relate a history of genital lesions in the past, or more specifically, during the latter part of pregnancy. A history of genital herpes in the mother's male consort(s) during her pregnancy is also helpful. Since the majority of genital HSV infections in pregnant women are subclinical, definitive diagnosis requires cytologic or virologic confirmation.

Cytologic techniques rapidly establish a presumptive diagnosis of neonatal HSV infection. Cell scrapings from the base of skin vesicles, conjunctival, or oral lesions in the neonate or genital lesions in the mother may be fixed with alcohol and stained with Papanicolaou's, Wright's, or Giemsa's stains. Typical virus-induced morphologic changes, including multinucleated giant cells and intranuclear inclusions, can be identified.

Isolation of HSV from an affected neonate establishes the diagnosis definitively. Samples for virus isolation in tissue culture should be obtained from vesicular fluid, mouth, conjunctiva, and cerebrospinal fluid (CSF). Instructions for media inoculation and transport have been outlined above. Because HSV grows rapidly in tissue culture, a positive culture will be recognized in 24-72 hours. Culture results from multiple sites define the extent of neonatal infection, and therefore, the infant's prognosis.

Treatment and Outcome

Infants who develop neonatal HSV should be placed in strict isolation and managed in a tertiary care facility. One cannot predict whether skin, ocular, or oral infection will remain localized or progressively disseminate and affect the CNS or retina. However, 75 per cent of infants presenting with skin lesions alone progress to dissemination; untreated, 75 per cent of these infants die, and neurologic or ocular sequelae commonly occur in survivors.

Recent data suggest that adenine arabinoside (ara-A, vidarbine) improves the outcome of disseminated and isolated CNS infection; overall mortality rate decreases from 75 to 40 per cent. Despite therapy, 85 per cent of survivors of disseminated neonatal HSV infection manifest neurologic or ocular sequelae. However, vidarabine decreases morbidity in infants surviving localized CNS or mucocutaneous HSV. Overall, vidarabine appears therapeutically beneficial in neonatal HSV infection. Nevertheless, the extent of neonatal herpetic disease before therapy determines outcome, and the long term prognosis remains poor for any infant with symptomatic infection. Treatment must be improved. A new drug, acyclovir, seems promising and is presently undergoing clinical trials.

Management of Infants at Risk

If an infected mother delivers her infant vaginally or by delayed cesarean section (membranes ruptured more than four hours), symptomatic neonatal HSV infection develops in 50 per cent or more of the infants. These newborns require strict isolation (isolette isolation is *not* adequate) for 21 days or until discharge. Alternatively, mother and infant can room together in a single room with secretion-excretion precautions. The infant's initial workup includes sentinel viral cultures of the eye, mouth and CSF, a CBC, liver function test, and CNS examination. If suspicious CNS (lethargy, irritability, seizures), respiratory (distress, cyanosis), gastrointestinal (vomiting, poor feeding, jaundice), or mucotaneous (vesicles, mouth ulcers, keratoconjunctivitis) signs appear, these studies bear repeating and a chest x-ray is indicated. At-risk infants require hospital observation for up to two weeks and weekly outpatient followup thereafter until one month of age. If symptomatic neonatal HSV infection develops, prompt transfer to a regional center experienced in management and treatment of these infants is warranted.

In contrast, when an infected mother delivers by cesarean section prior to rupture of membranes, her infant incurs minimal risk of HSV infection. Although separation of the infant from the mother appears optimum on theoretical grounds, mothers apprised of the risk and taught protective measures can care for their infants. Although a recent study suggests that breast milk transmits HSV, this risk does not presently outweigh the benefits of nursing. If the mother practices good handwashing, she and the infant can occupy a single room (secretion-excretion precautions). If the infant returns to the nursery, strict isolation is required. Because of the small but definite risk of HSV infection, the infant demands close observation both in the hospital and on a weekly outpatient basis until one month of age.

Infants infrequently acquire HSV from sources other than the maternal genital tract. However, case reports suggest that the mother, family members, and hospital personnel with recurrent herpes labialis (cold sores) can infect a newborn postnatally. This risk is a low, but potentially fatal one. Hospital personnel, patients, friends, and relatives with active HSV infection (cold sores, gingivostomatitis, herpetic whitlow) should not have contact with infants until their lesions crust. A mother who manifests an herpetic lesion at delivery or immediately postpartum can care for her infant if she appreciates the infant's risk and complies with protective measures. Before she handles her infant, she must scrupulously wash her hands and cover (by mask or gown) exposed active lesions. Unless an herpetic lesion affects the breast or surrounding skin, nursing is acceptable. When the mother strictly follows these recommendations, rooming-in effectively segregates the mother and infant (secretion-excretion precautions). If the infant returns to the nursery for any reason, strict isolation is required.

Because management and isolation guidelines for HSV infections are both controversial and in flux, consultation is available from Cheng Cho, M.D., Department of Pediatrics (913-588-6336) or Sterling B. Williams, M.D., Department of OB/GYN (913-588-6242), UKSM-KC; or Russell Nelson, M.D. (neonatal) or John Evans, M.D. (maternal), Division of Perinatal Medicine, Wesley Medical Center/ UKSM-Wichita (316-688-2360).

Acknowledgement

Patrick Hays, Ph.D., provided expert technical advice in the preparation of this material.

DIS-EASE

by Louis Forster, M.D., Salina

It was she
who suckled her fingers in secret
listening to my ongoing lectures all
medical students undergo: first
had been hearts and lungs and eyes.

Depression and psychosis.
Then babies.
Seeds and wombs and stages of labor appeared
a sort of circle to her.

I remember
they said she was good at what she did

and remember her
sitting in the lounge
with a pot of cold coffee after she
assisted me deliver a baby born early and bad
& drunk that died

and was there
when she overheard another doctor say
for three hundred years
infants have been conceived in gin
& incubated
in the product of low land barley and berries
& born
gingerly sometimes thwarting tears
of eyes dull and rolling
as dolls.

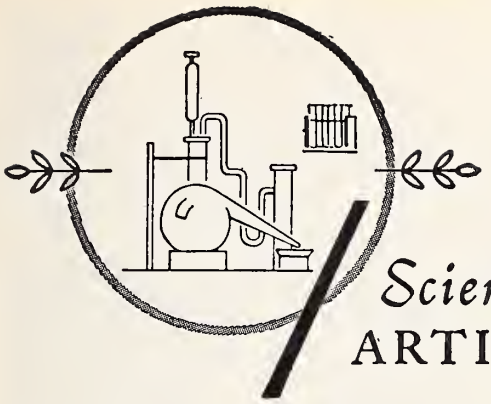
And I read
on the bulletin board her card about a

rubber gherkin
—guaranteed vatted three seasons—
she dedicates
to a Dutch doctor Fransiscus Sylvius
who gave gin birth
in 1652.

And like fading
light, saw her flicker, dip reappear still
holding tears taking shallow
gasps of breath rubbing her raw fingers

despite my arguing about
the commonness of the disease.

Ed. Note: Kansas Physicians have many talents aside from their professional efforts. This contribution by Dr. Forster introduces an innovation for the *Journal* but is also an invitation. It is hoped that this will inspire others to offer both prose and poetry for consideration of publication and perhaps, if the response warranted, the establishment of a continuing section in the *Journal*.



Arterial Trauma

Traumatic Nonpenetrating Injuries of the Cervical Internal Carotid and Vertebral Arteries

SOLOMON BATNITZKY, M.D. *and* **HILTON I. PRICE, M.D.,** *Kansas City, Kansas*

DAMAGE to the cervical segment of the internal carotid artery (ICA) or the vertebral artery as a result of blunt or non-penetrating injuries to the head and neck is uncommon, but does occur. ICA injuries may produce neurological deficits that mimic the signs and symptoms of acute cranio-cerebral injury. The morbidity and mortality rates associated with these injuries are alarmingly high. Vertebral artery injuries are less common and may produce varying degrees of neurological deficit involving the vertebrobasilar system. The morbidity and mortality rates of this latter group are less than those seen with carotid artery injuries, but they can also be significant.

Computed tomography (CT) has virtually replaced angiography as the definitive radiological procedure in the diagnosis and management of craniocerebral trauma. An arterial injury may go unrecognized because of the failure to suspect these injuries. Angiography is still the procedure of choice to demonstrate the vascular sequelae of head and neck trauma.

Physicians dealing with traumatized patients should be aware of this possibility and should not attribute all neurological signs in the traumatized patient to direct cerebral injury.

From the Department of Diagnostic Radiology, The University of Kansas School of Medicine-Kansas City.

Address reprint requests to Dr. Batnitzky, Dept. of Diagnostic Radiology, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.

Clinical Material

Presented here are 21 cases of injuries to the cervical segment of the ICA and three cases of vertebral artery injury following blunt or nonpenetrating trauma. In the ICA group, patients ranged in age from 16 to 57 years. The mean age was 28.2 years. There were 12 males and nine females in the series. The sources of trauma were: vehicular accidents, 18; head caught in metal punch press, one; fall on head (during seizure), one; and hit on neck by a chair, one.

Blunt or nonpenetrating trauma to the head and neck may on rare occasions result in damage to the cervical segment of the internal carotid artery or the vertebral artery. Mortality and morbidity rates associated with the former are alarmingly high. Mortality and morbidity rates for the latter are much lower, but can be significant. Angiography is still the definitive procedure to diagnose these lesions.

In 15 cases there was evidence of trauma to the forehead and face consisting of abrasions, contusions, and lacerations. In two cases there was external evidence of trauma to the neck, consisting of abrasions and contusions. Four patients exhibited no external evidence of trauma to the head or neck.

In 11 cases the onset of symptoms was immediate. In ten cases there was a delay of three hours to four

days in onset of the neurological symptoms.

The three patients who presented with vertebral artery injury were a 9.5-month-old male, a 30-year-old female, and a 57-year-old male. Their injuries occurred following spontaneous rotation of the head; hyperextension and rotation of the head and neck while sliding into third base during a soft ball game; and — in the third case — chiropractic manipulation. In all three cases, there was no external evidence of trauma to the head or neck. The onset of posterior fossa signs and symptoms occurred immediately in the infant and after a latent period of six and nine hours in the other two patients, respectively.

Roentgenographic Investigation: ICA

Plain skull films were normal in all 24 patients. Two patients had fractures involving the angle of the mandible, and one patient had a depressed fracture of the sternum. Cranial CT was performed in nine patients on admission to the hospital and was repeated in five of these patients following neurological deterioration which they experienced 3-24 hours later.

In two patients, the initial CT scan demonstrated diffuse bilateral cerebral edema. The CT scans in the remaining seven patients and those five patients who had followup CT scans following neurologic deterioration were normal.

Bilateral carotid angiography was performed in these 21 patients. Bilateral involvement of the cervical ICAs was demonstrated in five patients *(Figure 1)*. Of these five patients, four had bilateral dissections but without total occlusion of the artery. The other patient had a dissection on one side and complete occlusion on the opposite side. One of the patients with bilateral dissecting aneurysms of the ICA exhibited an associated traumatic aneurysm on one side. Fourteen patients had complete occlusion of one ICA *(Figure 2),* and two patients showed evidence of dissection of one carotid artery without total occlusion *(Figure 3)*. In those cases in which complete occlusion of the cervical ICA was demon-



Figure 1a. Right AP carotid angiography in a 25-year-old female who was involved in an automobile accident demonstrates a dissecting aneurysm (arrowheads) involving the cervical segment of the ICA.



Figure 1b. Left AP carotid angiography in the same patient demonstrates similar changes involving the cervical segment of the left ICA (arrowheads).

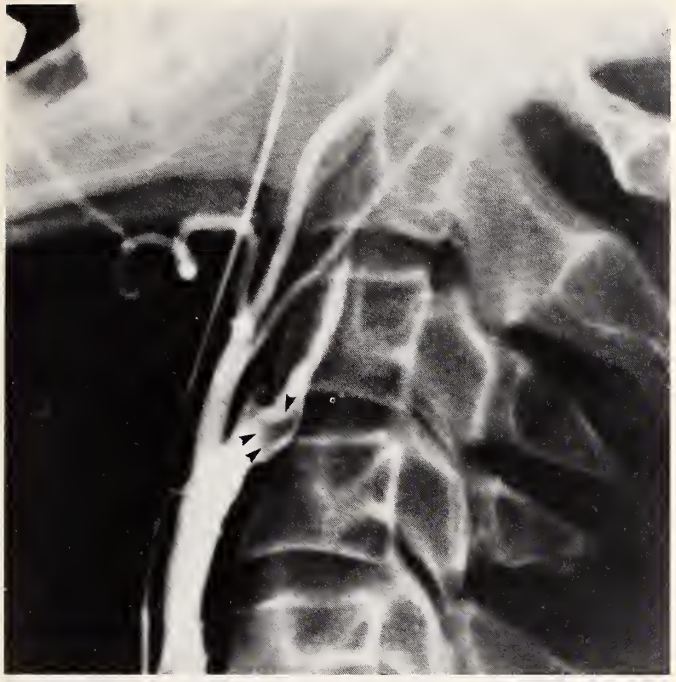


Figure 2. Right lateral carotid angiography in a 42-year-old male auto accident victim demonstrates tapering and complete occlusion of the proximal 3 cm of the ICA. Thrombus (arrowheads) can be seen within the contrast filled proximal segment.

strated, the site of occlusion occurred within the first 3 cm of the proximal ICA.

Roentgenographic Investigation: Vertebral Artery

Plain skull films were normal in all three patients. Cervical spine films were normal in two patients. In the patient who had chiropractic manipulation, cervical spine films demonstrated generalized osteoarthritic changes.

CT head scans were performed in all three patients on admission to the hospital. In two cases, posterior fossa infarcts were demonstrated. These scans were performed 30 hours and nine days, respectively, after injury *(Figure 4)*. In the third case, the CT scan, performed six hours after injury, was normal.

Vertebral angiography in two patients demonstrated occlusion of one of the vertebral arteries at the level of C1-C2 *(Figure 5)*. Digital subtraction angiography in the patient who had chiropractic manipulation demonstrated occlusion of the right vertebral artery at its origin *(Figure 4)*.

Clinical Outcome

In the ICA group of patients, nine underwent surgery consisting of arteriotomy with thrombectomy or thromboendarterectomy. Of these nine patients, two died shortly after surgery. One patient improved without any residual neurological deficit.



Figure 3. Lateral right carotid angiography in a 27-year-old female auto accident victim demonstrates irregular narrowing of the lumen of the upper cervical ICA due to a dissecting lesion.

Six patients improved slightly but still had significant residual neurological deficits at the time of discharge and followup up to one year post-injury. In the group of 12 patients who did not have surgery, five died. Six were left with a moderate to severe neurological deficit, and one patient recovered completely.

Autopsies were performed on five of the patients who died. (Four were from the group who did not have surgery.) In all five cases, there was evidence of one or more intimal tears of the cervical ICA at the level of C1 and C2. Extensive intraluminal antemortem thrombus was present in all five cases, and three cases showed evidence of intramural dissection.

In the vertebral artery group, the two adult patients were treated conservatively with anticoagulant therapy. Their neurological deficits gradually resolved and disappeared during a period of three to four months. The 9.5-month-old-boy, who was the subject of a previous communication,[1] developed massive cerebellar infarction and acute hydrocepha-

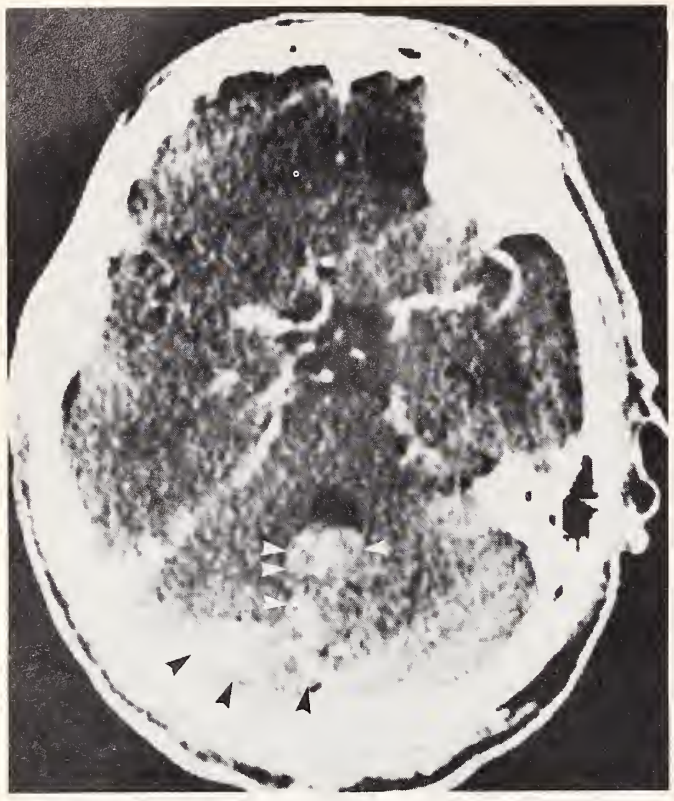


Figure 4a. Post-contrast CT head scan in a 57-year-old male demonstrates right-sided cerebellar infarction (arrowheads). This patient underwent chiropractic manipulation nine days previously.



Figure 4b. Digital subtraction angiography in the same patient demonstrates complete occlusion of the right vertebral artery at its origin (arrowheads).

lus. This was treated by ventricular drainage. The patient's neurological deficits resolved completely during the next few months and at four-year follow-up, the patient was completely asymptomatic.

Discussion: ICA

Traumatic ICA occlusion following blunt trauma was first reported by Verneuil more than 100 years ago.[2] Reports of post-traumatic changes in these vessels have been relatively frequent since the beginning of the automobile age.[3-5] In the overwhelming majority of cases, trauma to the ICA is the result of penetrating injuries to the vessel. Nonpenetrating or blunt injuries of the cervical ICA account for only 3-10 per cent of all cervical carotid artery trauma.[4, 5] Nonpenetrating or blunt trauma to the cervical ICA may result in partial or complete thrombotic occlusion or aneurysm formation. These injuries are associated with a high incidence of severe neurologic complications and significant mortality.[3]

Pathogenesis and Mechanisms of Injury

Non-penetrating or blunt trauma to the cervical ICA may be caused by indirect trauma due to hyperextension of the neck; direct blunt trauma to the neck; intra-oral trauma; or fractures through the base of the skull.

Hyperextension Injuries. Closed head trauma, particularly in auto accidents, is often accompanied by forced hyperextension of the head and neck. The ICA is tensioned and stretched over the bony mass of the first and second cervical vertebrae, tearing the intima in one or more places.[6-10] In some cases, the inner layer of the media may also be torn. This is the probable mechanism of injury in the majority of cases. In reviewing 52 cases in the literature, Yamada *et al.*[3] found an intimal tear in 62 per cent of cases in which the pathology was definitely known.

Sudden stretching of the femoral artery has been shown experimentally to produce an intimal tear.[11] This is followed by rolling up of the ruptured intimal layer by the flow of the blood stream. Thrombosis at the site of the intimal rupture results in occlusion of the artery. These changes are similar to the changes that occur in the carotid artery with hyperextension.

The presence of lacerations, contusions, and abrasions on the forehead, face, and jaw in many of the



Figure 5. Left vertebral angiography in a 30-year-old female demonstrates complete occlusion of the vertebral artery at the atlantoaxial level (arrowheads).

reported cases in the literature[8, 10] and in 15 of our cases, indicates that hyperextension of the head occurred at the time of injury.

Contralateral rotation of the head at the time of injury may cause further stretching and compression of the ICA.[7, 8, 10] The combination of hyperextension and contralateral rotation of the head may be the mechanism involved in unilateral ICA trauma. However, the bilateral nature in five of our cases and in other reported cases in the literature[9, 12] argues against the rotational element as a major factor in the production of the intimal tear.

Boldrey *et al.*[6] postulated fixation of the ICA to the lateral process of the atlas as a result of previous pharyngeal infections. This could increase the probability of injury at this level from sudden and severe hyperextension of the head.

One patient in our series who had a total occlusion of the right cervical ICA had an associated fracture of the sternum. Hughes and Brownell[7] showed that depressed sternal fractures may tether the innominate artery so that the distal common carotid artery and ICA are more susceptible to sudden stress injuries. This may play a role in some patients.

The intimal tear may act as a nidus for thrombus formation.[10] There may be antegrade or retrograde propagation of the thrombus. In the majority of cases, total occlusion of the ICA is present;[3] this occurred in 15 of our 21 patients. The thrombus may also serve as a source of emboli. The release of tiny emboli from the ICA thrombus into the cerebral circulation may result in neurologic deterioration.

Less commonly, a dissecting aneurysm may develop.[8, 9] Passage of blood through the intimal tear into the media may result in intramural dissection with a resultant narrowing and compromise of the vascular lumen. The resultant stenosis of the lumen may lead to thrombotic occlusion of the vessel and the complications of emboli.

The presence of perivascular edema or hematoma, vasospasm, hypotension, and the increased adhesiveness of platelets following major trauma increases the tendency for thrombus formation.[10]

The most common site of ICA injuries is the upper cervical internal carotid artery at the level of C1 and C2.[3, 8, 9] There have been a number of reports that suggest that the most frequent site of injury is the cervical ICA 1-3 cm above the bifurcation of the common carotid artery.[10, 13] However, the proximal propagation of the thrombus from the upper cervical ICA toward the bifurcation of the common carotid artery may lead to the erroneous impression that the site of arterial injury is nearer the bifurcation of the common carotid artery.

Cervical ICA aneurysms due to blunt trauma are extremely rare but may occur if there is disruption of the media or the adventitia as well as the intima at the time of injury.[9] Rupture of these aneurysms is rare, but complications secondary to embolism and thrombosis are relatively common.

Direct Trauma. Direct blows to the neck, particularly in older patients, are more likely to damage an atheromatous artery with dislodgement of the atheromatous plaque which may form an obstructing flap with associated thrombus formation.[3, 14] Two patients in our series had external evidence of trauma to the neck on the affected side. One of these patients, aged 57 years, had a history of hypertension and diabetes. Angiography in this patient, in addition to the occlusion of the cervical ICA on the side of the neck abrasion, demonstrated generalized atherosclerotic changes in the ipsilateral common carotid artery, the contralateral common carotid bifurcation, the aorta, and the femoral arteries. Even more rarely, blunt trauma to the neck may cause bleeding into the soft tissues around the carotid arteries, and this

may produce extrinsic constriction and occlusion of the carotid artery.[15]

Batzdorf[9] has shown that forces applied to the angle of a mandible which may or may not fracture the mandible itself can be transmitted to the ICA or could compress the artery against the cervical spine and thus injure the artery. Two patients in our series with unilateral total occlusion of the cervical ICA had fractured mandibles on the same side as the arterial occlusion.

Intraoral Trauma. Thrombosis of the ICA resulting from intraoral trauma to the peritonsilar area has been reported in children who have fallen while sucking on a hard object such as a lollipop, pencil, or stick.[16]

Basal Skull Fractures. These fractures may contuse or tear the petrous portion of the internal carotid artery which may, in turn, result in thrombosis.[3, 13]

Clinical Features. These lesions occur mainly in young patients. The mean age in our series was 28.2 years. The greater mobility of the cervical spine in younger individuals may account for the increased frequency of this type of injury in patients under 50 years of age.[9] Furthermore, in older patients, the vessels lose their elasticity and become tortuous and hardened. This makes the vessels more resistant to tearing and diminishes the possibility of a stretch injury to the ICA.

The clinical course and features of traumatic injury to the cervical ICA vary considerably from patient to patient.

The prognosis following injury of the ICA depends on the adequacy of collateral circulation and the presence or absence of embolic phenomena.

Because of adequate collateral circulation from the contralateral carotid artery, total occlusion of a single carotid artery may be well-tolerated in most young individuals. However, the patient may deteriorate neurologically if complicating factors such as hypoxia, hypotension, distal propagation of the clot, or embolization into the intracranial vessels occurs.

The development of symptoms and signs may follow a lucid interval varying from one to more than 24 hours after the trauma, thus simulating an epidural hematoma. This may direct the physician's attention to the cranium as the cause for the neurological deficit.

There are instances where the latent period between the time of injury and the development of the clinical picture may extend to a few days or even a few weeks. The clinical picture may be aggravated by associated intracranial injuries. Bilateral cerebral edema was present in two of our patients.

The development of focal neurologic deficits with essentially no change in the level of consciousness suggests a post-traumatic vascular lesion and may be an important sign to differentiate a vascular injury from an intracranial mass lesion.[5, 17] Patients with traumatic intracranial mass lesions generally become obtunded by the time focal neurologic signs appear. However, this was not a feature in our ten patients who deteriorated after a latent period. In all ten patients, mental blunting accompanied the development of focal neurologic deficits.

Yamada and others[3, 9] have shown that there is a high incidence of morbidity and mortality if surgical treatment consisting of thromboendarterectomy is not prompt once neurological signs have appeared. There was a 14 per cent mortality rate in the operated group that he reviewed (21 cases), compared with a 55 per cent mortality rate in the non-operated group (31 cases). In the survivors of the operated group, 10 per cent were left with moderate or slight neurological deficits, while 18 per cent had no neurological deficits. The survivors in the non-operated group were so disabled as to be unable to work.

The mortality and morbidity rates in our series are similar to those reported in other series.[3, 9]

Discussion: Vertebral Artery Injuries

The cervical ICAs are more subject to trauma because of their relatively unprotected location in the soft tissues of the neck, than the cervical segment of each vertebral artery. The vertebral artery is protected by the rigid bony rings of the foramina transversaria through which the vertebral artery travels up the cervical spine from the sixth to the first vertebra.

Whereas the overwhelming majority of cervical ICA trauma is caused by penetrating injuries, most vertebral artery injuries are secondary to blunt trauma.[18]

Partial or complete occlusion of the vertebral artery may occur following chiropractic manipulations of the neck, yoga exercises, calisthenics, football injuries, and bow hunting.[18]

Vertebral artery injuries may result in varying degrees of neurological deficit involving the vertebrobasilar system.

Although predisposing factors such as non-fusion of the odontoid process with atlanto-axial subluxation, cervical osteoarthritis, fracture or dislocation of the cervical spine and atherosclerotic vascular disease have been described in association with these injuries, in many patients no underlying disease can be found.[1] This was true in two of our three patients. In the third patient, generalized cervical osteo-arthritis was present.

Rotation or hyperextension of the head and neck are by far the most significant modes of injury to the vertebral arteries.[1, 18, 19] At the atlanto-axial joint, the vertebral artery can be narrowed or even occluded during rotation of the head to the opposite side.[19]

Although in most cases reported in the literature the vertebral artery injury produced is due to a major traumatic event, occlusion of a vertebral artery has been documented after mild traumatic events.[1, 19] This occurred in two of our patients. In fact, in the 9.5-month-old boy, the injury was precipitated by spontaneous rotation of the head.

Rotation of the head with or without hyperextension may cause spasm or an intimal tear of the vertebral artery at the level of the atlanto-axial joint.[1, 20] This may result in thrombosis with or without intramural dissection of the artery. Embolization into the vertebrobasilar system may result. Rarely, a traumatic pseudoaneurysm of the vertebral artery at this level may occur.[20]

The risk of vertebral artery injury by rotation or hyperextension seems to increase in the presence of a fracture or dislocation of the cervical spine, atherosclerosis, or osteoarthritis.[19] The site of injury to the vertebral artery in such cases will be determined by the underlying pathological state. This was true in the patient who underwent chiropractic manipulation and who had generalized cervical osteoarthritis. The site of occlusion of the vertebral artery was at the origin of the artery. In this patient, it is possible that the initial injury may have been at a higher level, but by the time angiography was performed, proximal propagation of the clot had occurred.

Our three patients recovered completely from their neurological deficits. However, reports in the literature[18] have shown that these lesions can be associated with significant morbidity and mortality.

Roentgenographic Features

Initially, CT is relatively ineffective in the diagnosis of the sequelae of carotid and vertebral artery trauma. However, delayed serial CT scans may demonstrate the evolution of infarction in these cases. In French *et al.*'s series,[17] the earliest positive scan to show infarction occurred 11 hours after the onset of neurologic deficits. No CT scan performed within 24 hours after the onset of symptoms was positive in our series.

Carotid angiography is the definitive radiologic procedure to demonstrate ICA trauma. Bilateral carotid angiography, to include both the neck and intracranial vessels, should be performed in all cases of suspected ICA trauma. Angiography will demonstrate the arterial injury and sites of cerebral emboli, and will evaluate collateral circulation. If the clinical situation suggests posterior fossa involvement, vertebral angiography should also be performed.

In ICA trauma, angiography in the majority of cases will demonstrate total occlusion of the cervical ICA 1-3 cm distal to the bifurcation of the ICA. In other cases, varying degrees of stenosis of the artery may be seen. The intimal tear or flap may be seen as a linear or spiral lucency within the opacified vessel.

The characteristic features of a dissecting aneurysm consisting of marked irregularity and narrowing of the arterial lumen may be seen. If the false lumen of the dissection joins the normal lumen of the vessel at both ends of the lesion, angiography may opacify both the true and false lumens of the vessel. The intima and that portion of the tunica media central to the dissection will appear as a spiral band-like filling defect between the two lumens.

Rarely, a false aneurysm may be demonstrated, typically in the upper cervical segment of the ICA.

Similar angiographic changes may be seen in vertebral artery trauma. In the majority of cases, the site of injury is in the region of the atlanto-axial joint.

Conclusion

An arterial injury should be considered in the differential diagnosis of craniocerebral trauma. These lesions are rarely suspected clinically, as in most instances there may be no external evidence of trauma to the neck. The possibility of injury to these vessels should be considered in any patient who after a latent interval following injury, whether severe or minor, begins to develop neurological signs or symptoms. The vascular sequelae of head and neck trauma may be missed if one relies only on CT. Angiography is the definitive procedure of choice in diagnosing these lesions.

Prompt and early diagnosis is essential to improve the bad prognosis associated with these lesions.

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"Untreatable" Neuromuscular Disease

Treatment Trials and Symptomatic Treatment of Amyotrophic Lateral Sclerosis

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RENEWED INTEREST in the etiology and pathogenesis of amyotrophic lateral sclerosis (ALS) has developed in recent years. Several recent medical symposia have documented the progress in our understanding of this enigmatic disease. Several of these symposia have focused on possible research approaches in ALS; the most comprehensive was that sponsored by the Amyotrophic Lateral Sclerosis Society of America (ALSSOA) in 1975.[1] A clinically oriented symposium, again sponsored by ALSSOA, was held in 1979 and resulted in the publication of a book entitled *The Diagnosis and Treatment of Amyotrophic Lateral Sclerosis*.[2] Recently, symposia on the Pathogenesis of Human Motor Neuron Diseases in 1981, sponsored by the Muscular Dystrophy Association, and in 1982, Progress in Motor Neuron Disease sponsored by MND Association of Great Britain were held. Proceedings of these are currently in press.

In reviewing the previous approaches to etiology and pathogenesis we found that the focus has been directed toward uncovering the single cause of ALS.[3] This approach becomes misguided if one considers that ALS might represent a syndrome of the neuromuscular system produced by a number of different causes.[4-6]

In a review of past therapeutic trials in ALS. Festoff and Crigger[3] considered the various theories concerning the etiology of ALS *(Table I)*. Almost all of these studies were totally inadequate to rule out a particular form of therapy, and hence the etiologic theory it was directed toward, or to retain it for another trial. With so many potentially still viable "theories" concerning etiology, the probability that ALS is a syndrome resulting from effects of various external or internal stimuli becomes even greater.[4-6] Festoff[7] hypothesized that such stimuli might become manifest through a common pathogenetic mechanism; namely, the activation of extracellular neutral protease activity, beginning at the neuromuscular junctions, and extending along the length of the skeletal muscle fiber *(Figure 1)*. This hypothesis was compatible with the concept of "dying-back" as it related to ALS.[8, 9] Also suggested in the hypothesis by Festoff[7] was extension of enhanced protease activity to the central nervous system. This latter suggestion, coupled with the loss of peripheral target organ and retrograde transynaptic degeneration might account for the corticospinal tract degeneration as well as the motor unit disturbance in ALS. The dying-back model, in this context, becomes progressive destruction of nerve-muscle adhesion and the degradation of the basal lamina (BL) in the neuromuscular junction.

Results of therapeutic trials indicate that amyotrophic lateral sclerosis may be a syndrome resulting from effects of various external or internal stimuli. Extensive compilation of the results of these trials have led to an hypothesis, presented here, that increased activity of extracellular proteases in muscle connective tissue and at the neuromuscular junction initiates and continues nerve-muscle degeneration.

Basal Lamina and Neuromuscular Interaction

A layer of BL of 80-100 nm thick ensheathes the adult skeletal muscle fiber and contributes a considerable amount of the synaptic matrix material at the neuromuscular junction (NMJ). This BL may play important roles in the formation, function and maintenance of the NMJ, as shown by a number of studies in recent years *(Table II)*.

Characterized cytochemically[10, 11] but not biochemically, biological studies suggest that it may be possible to characterize and isolate BL-associated

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TABLE I
THEORIES OF ETIOPATHOGENESIS IN ALS

Deficiency of a "factor"
Vitamin E
Vitamin B_{12}
Environmental "factor"
Heavy metals — lead, mercury
Selenium, calcium, manganese, aluminum
Cycad (Guamanian ALS)
Metabolic "factor"
Glucose and insulin metabolism/endocrine dysfunction
Exocrine dysfunction — pancreas, salivary glands
Catecholamine metabolism (HVA and 5-HIAA)
Abnormal mineral metabolism — hyperparathyroidism
Free amino acids
Aging "factor"
Collagenase and proteases
Viral "factor"
Cancer "factor"
Paraneoplastic syndrome
Circulating "factor"
Cytotoxicity of sera

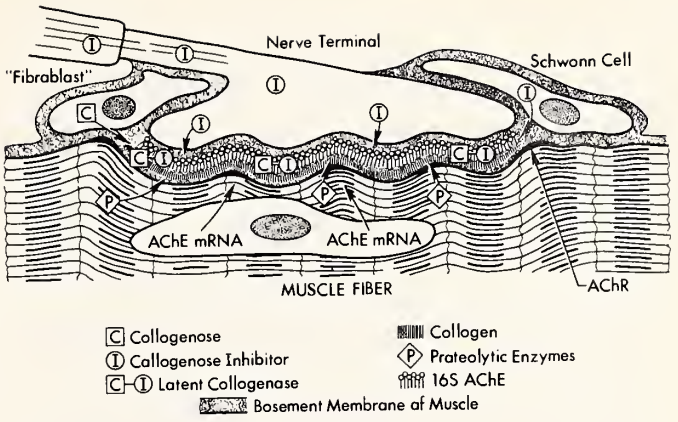


Figure 1. Schematic view of mammalian neuromuscular junction showing nerve terminal, "fibroblast," Schwann cell, and muscle fiber, AChE is depicted as being synthesized in muscle (although clearly nerve also synthesizes this enzyme being secreted and interacting with both collagen and some molecule of the basement membrane (fibronectin?)). Collagenase is secreted by "fibroblasts" but kept inactive by inhibitors secreted by Schwann cells, nerve terminal, or both. Acetylcholine receptor (AChR) is shown concentrated on muscle plasmolemma at the peaks of secondary folds.[7]

macromolecules.[12] Changes in BL-associated macromolecules may occur in ALS similar to those seen in glomeruli of diabetic patients.

Extracellular Proteases and NM Function

A number of studies by our laboratory suggest that proteases may be involved in changes of several BL-associated muscle proteins: rapid decay of 16S endplate acetylcholinesterase (AChE),[13, 14] fibronectin[15] and other BL macromolecules in studies of experimental denervation. Such proteases may be activated by nerve or muscle injury. They may be

TABLE II
ROLES OF BASAL LAMINA (BL) AT NMJ

Role	*References*
1. Synaptic BL regulates nerve terminal differentiation during reinervation	1. Sanes *et al.* (1978) *J. Cell Biol* 78:176
2. Synaptic BL may induce/maintain clustering	2. Burden *et al.* (1979) *J. Cell Biol* 82:412
3. Synaptic BL connected via sites to A_{12} EP-specific AChE form	3. Betz and Sakman (1973) *J. Physiol.* 230:673 Festoff and Engel (1974) PNAS 71:2435 Hall and Kelly (1971) *Nature New Biol* 232:62 McMahan *et al.* (1978) *Nature* 271:172 Fernandez *et al.* (1979) *J. Neurobiol.* 10:441 McLaughlin *et al.* (1977) *J. Neurochem* 31:783
4. Adhesion of nerve to muscle depends on integrity of BL	4. Festoff *et al.* (1977) *J. Membrane Biol.* 32:345
5. Synapse specific BL components at NMJ suggest contribution by nerve and muscle; become specialized after AChRs cluster and before AChE	5. Sanes and Hall (1979) *J. Cell Biol.* 83:357
6. Therefore, synaptic BL plays important roles in formation, function and maintenance of NMJ implying role in regeneration	6. All above references

TABLE III
MECHANISMS CONTROLLING PROTEINASES*

Proteinases only present as inactive precursors in human plasma (*i.e.*, plasminogen)
Activators (*i.e.*, plasminogen activators) frequently separated from proteinases by cell barrier
Activators liberated only when protease activation biologically required (*i.e.*, tissue remodeling)
Activation is local, controlled by inhibitors to both activators and proteinases (*i.e.*, anti-thrombin III, α_2-macroglobulin, alpha$_1$-antitrypsin)
Protease inhibitors (PIs) not only in plasma but in proteolytic-sensitive organs and tissues such as mucous membrane of respiratory and gastro-intestinal tract, muscle, neuromuscular junction, and central nervous system

* As modified from Heimburger (1975).

produced by muscle or other non-muscle cells such as mast cells.

Extracellularly-acting proteases and bacterial collagenase have been shown to affect BL-bound AChE at NMJ.[16-18] Collagenase, elastase, and plasminogen activators (PAs) besides acting extracellularly, have pH optima in the neutral range, thus making them reasonable mediators of degradative activity on NMJ macromolecules. PAs' possible involvement in nerve-muscle interaction at the NMJ is suggested by the recent demonstration that Schwann cells wrap neurites *in vitro* and migrate under specified culture conditions as a function of the plasmin they generate and release into the medium.[19] Plasminogen is present in abundant quantities in plasma (0.1 mg/ml), and many cells appear capable of synthesizing and releasing PAs thereby generating plasmin which has broad specificity.[20] Control over excessive proteolysis is mediated by circulating antiproteases (proteins capable of inhibiting plasmin, collagenase, elastase and other proteases): alpha$_2$-macroglobulin, alpha$_1$-antitrypsin, C_1-inactivator and others[21] *(Table III)*.

The balance of this system depends partly on the protease inhibitors (PI) concentration in plasma and partly on the local concentration of PAs synthesized and released. Collagenase, shown to exist in a latent or "zymogen" form, can also be activated by these and other proteases by specific proteolytic cleavage or perhaps by plasmin itself.[22] The model recently proposed[7] attempts to account for changes that occur at NMJ in experimental denervation, including release of 16S AChE, and might also be involved in the pathogenetic basis for ALS *(Figure 1)* in terms of activation of such proteases. Recently, our group has identified and characterized a PA in cultured skeletal muscle cells.[23] A similar protease is concentrated in and released by denervated muscle in organ culture (Festoff, in preparation).

TABLE IV
COMPARISON OF ALS ETIOLOGIC THEORIES AND PROTEASE ACTIVATION

Theory	*Activates Neutral Proteases*
Deficiency	Unknown
Environmental	
Lead	Yes
Mercury	Yes
Manganese	Unknown
Calcium	Yes
Aluminum	Unknown
Metabolic	
Glucose/insulin	Yes
Pancreatic	Yes
Catecholamine	Unknown
Free amino acids	Yes
Aging	Yes
Viral	Yes
Cancer	Yes
Circulating	Possibly (*i.e.*, plasminogen)

Proteases in Denervation

Generalized proteolysis increases in skeletal muscle following denervation.[24] The earliest increase in total muscle proteolytic enzymes following nerve transection is between 24-48 hours.[25] In addition to denervation, increased proteolysis has been found in a number of neuromuscular disorders.[26, 27] Activation of collagenase, endogenous inhibitors of collagenolytic activity and other factors involved in collagen resorption, have been implicated in arthritis, but similar information is unavailable for denervating or other neuromuscular conditions. What role such proteases have in ALS, where arthritic complaints are common, remains speculative. However, the previous finding that skin of patients with ALS and other denervating disorders possessed increased collagenolytic activity[28, 29] suggests a connection between dependence of neurotrophic control of muscle BL metabolism and enhanced proteolytic and collagenolytic activity in ALS tissue.

Treatment Trials

As discussed previously by Festoff and Crigger,[3] the history of therapeutic trials in ALS is confusing at best. Of interest, however is the striking congruity between the proposed etiologic theories of ALS and those phenomena known to activate neutral protease activity *(Table IV)*.

Following this line of reasoning one would ideally

TABLE V

Action	*Drug*
MUSCLE CRAMPS	
Muscle membrane stabilizers	Phenytoin (Dilantin), 200-400 mg/day Quinine Sulfate, 180-300 mg/day
SPASTICITY AND FLEXOR SPASMS	
Derivative of inhibitory neurotransmitter GABA, increases inhibition by interfering with the release of excitatory transmitters	Baclofen (Lioresal, 5-25 mg QID
Facilitates inhibition mediated by GABA	Diazepam (Valium), 2-10 mg QID

GABA (gamma amino isobutyric acid).

TABLE VI
TREATMENT OF SIALORRHEA

Due to decreased bulbar control and, possibly, increased salivary gland output (autonomic involvement?).
Amitryptiline (Elavil) 50-100 mg daily in divided doses. Dual action: increases mood and decreases saliva due to anti-cholinergic effect.
Clonidine (Catapres) 0.1-0.2 mg daily decreases salivary output due to blocking central sympathetic outflow.
Best to avoid atropine because of tendency to cause inspissation of mucous and plug formation.
Transtympanic neurectomy useful in some patients — secondary hypertrophy minor palatal salivary glands prevents excessive dryness.

seek to initiate therapeutic trials with agents to augment naturally-occurring protease inhibitors. Alternatively, one would want to evaluate exogenous agents in order to directly inhibit protease activity. Encouraging results in this regard have already been obtained in experimental animals. Leupeptin, a tripeptide inhibitor of both lysosomal and neutral proteases,[30] reverses the muscle atrophy found in murine dystrophy[31] and following denervation[32] as well as the degeneration seen in cultured muscle.[33] We and others have shown that leupeptin also slows the turn-over of acetylcholine receptors (AChR) in cultured muscle.[34-35] Similarly leupeptin, pepstatin and aprotinin (Trasylol) prevent the rapid loss of 16S AChE following denervation.[36] The latter study supports the notion that denervation-induced nerve-muscle junction degeneration is initiated by proteolytic attack on superficially attached proteins such as 16S AChE.[7, 13] A further implication, one currently being tested in our laboratory, is that nerve fibers may contain one or more factors to inhibit or suppress muscle protease activity.

Neutral protease inhibitors such as epsilon amino-caproic acid (Amicar), aprotinin (Trasylol), and leupeptin are all effective in inhibiting the PA of cultured skeletal muscle cells.[23] If plasminogen could gain access to the NMJ itself, PA can convert it to plasmin, and α_2-macroglobulin or other plasma inhibitor would be required to control such activity. Inadequate inhibitor or too much protease would result in continued degradation. Exogenous inhibitors could positively affect this imbalance.

In our ALS Regional Research Center and the MDA Clinic at KUMC, eight patients with classical ALS have received up to 20 grams daily (oral doses) of Amicar in an open trial. Our preliminary results suggest stabilization in three patients but the drug was discontinued in four patients because of gastrointestinal side effects and in one who developed urticaria. A therapeutic trial with intravenous aprotinin (Trasylol) has received FDA approval and has just been initiated.

Symptomatic Therapy

In the absence of specific therapy certain treatment modalities are available to help patients at different stages of the disease. Physical therapy and exercise programs can be tailored to the individual's needs. Excellent reviews on these subjects are available and may be consulted.[37] Five major areas, outside of specific axo-appendicular weakness, need to be addressed sometime during the progressive course of the disease. They are: increased muscle tone and cramps; sialorrhea; dysphonia; dysphagia; dyspnea and respiratory distress. A team approach to these problems is essential. Such a team has been developed at KUMC and at the Kansas City Veterans Administration Medical Center.

Spasticity and Muscle Cramps

For the relief of spasticity and increased tone we regularly evaluate ALS patients for treatment with baclofen (Lioresal). This agent was previously evaluated for its usefulness in multiple sclerosis and spinal cord injuries,[38, 39] but varying reports of efficacy in severe upper motor neuron involvement in ALS have also appeared.[40, 41] Gradual increase in dosage to 60-80 mg daily (occasional patients require more) often improves coodination while walking as well as diminishing flexor spasms at night. In addition to baclofen, we also utilize other agents as

listed in *Table V*. Phenytoin (Dilantin) is also quite effective in reducing spasticity, muscle soreness, and cramps.[42]

Sialorrhea

Increased salivation can be debilitating during the course in ALS patients when bulbar involvement is prominent. Drooling occurs because of a combination of impairment in swallowing and weakness of tongue and facial muscles involving the lips. However, it is our impression that increase in salivary flow rates do occur in this disorder. Several medications diminish sialorrhea and should be considered *(Table VI)*. Amytryptiline (Elavil) 50-100 mg daily in divided doses, has central, as well as antidepressive and mild sedative, anticholinergic actions. Recently, we have begun to use protryptiline (Vivactil) because it is less sedating. If the use of tricyclics is contraindicated, trihexyphenidyl (Artane), 2 mg TID has equally powerful anticholinergic actions. Clonidine (Catapres) 0.1-0.2 mg daily decreases salivary output due to decreased central sympathetic outflow, but caution must be exercised regarding blood pressure lowering. As a last resort, bilateral transtympanic neurectomy of the chorda tympani nerve is feasible. After this procedure, excessive oral dryness is avoided by compensatory hypertrophy of the minor salivary glands. It should be kept in mind that anticholinesterase medication (*i.e.* pyridostigmine, Mestinon) can increase salivary secretion via direct muscarinic stimulation. If secretions in the oropharynx become thick and tenacious one papaya extract tablet or meat tenderizer (*i.e.*, Adolph's) sprinkled on the posterior tongue liquefies quite effectively.

Dysphonia and Dysarthria

ALS patients have reduced tidal air volume with diminished strength in exhalation. The vocal cords may be either flaccid or spastic, depending on the extent of lower *vs* upper motor neuron involvement. There may be velopharyngeal weakness permitting air coming from the larynx to be emitted through the nose rather than through the mouth. Articulation requires coordinated and adequate movement of tongue, lips, and mandible which are frequently affected by a combination of upper and lower motor neuron involvement in ALS.

Treatment is aimed at first training the patient to overcompensate for distorted articulation.[43] With early bulbar involvement the unintelligibility of nasal emission may be satisfactorily corrected with a palatal lift prosthesis. This device attaches to the upper teeth and projects an acrylic plate posteriorly that holds the soft palate in an elevated position. This partially closes the velopharyngeal port, thus preventing air escape. Teflon or silicon injection into the posterior pharyngeal wall theoretically serves the same purpose, although it has been less thoroughly tested. Agents that diminish spasticity (diazepam, baclofen) can benefit the pseudobulbar component of the dysarthria. See *Table VII*.

A mechanical ventilator (Pneumobelt) or cuirass respirator can add force to the exhalation to aid in speaking. A hand-held amplifier can also increase the volume of the voice. Electronic speech systems that store words or sentences are also available as are more complicated non-verbal communication devices. Our speech and language departments at both KUMC and KCVAMC have developed a keen interest in this area.

Dysphagia

When progressive bulbar involvement involves the swallowing mechanism, initially adjustments in the texture of food may suffice. Pureed food is swallowed more easily than solid food or clear liquids. A trial with anticholinesterase (*i.e.* Mestinon) before meals is warranted in all cases. Subsequently, a pediatric-size, nasogastric tube (*i.e.* Dobhoff) can be

TABLE VII
TREATMENT OF DYSPHONIA AND DYSARTHRIA

Muscle relaxants (*i.e.* Valium, Lioresal) may improve spastic speech problems
Use of palatal lifts early in bulbar involvement
No evidence that surgical treatment effective, but injection of teflon or silicone into nasopharynx might affect hypernasal quality
If due to alveolar hypoventilation, improvement may occur with mechanical ventilator

TABLE VIII
TREATMENT OF DYSPHAGIA

Oral anti-cholinesterase before meals
Neostigmine 30-45 min a.c.
Pyridostigmine (Mestinon) 1 to 1½ hr a.c. 30-60 mg, can use syrup
Use of pediatric size polyethylene NG tube (Dobbhoff)
Patients can place and remove just prior to feeding — tube feeding formula required
Surgical intervention
Cricopharyngeal myotomy — only after defined cricopharyngeal bar of spastic component
Cervical esophagostomy — most satisfactory, well tolerated — left side of neck — local anesthesia — fistula heals 2-3 weeks — can handle home-prepared blenderized food.

placed through the nose and left for relatively long periods of time provided that the patient and family are taught how to handle it. Cricopharyngeal myotomy may be advisable in cases when barium swallow cineradiography demonstrates a spastic contraction of this muscle.

The most satisfactory stomal surgical method is cervical esophagostomy. It is preferable to gastrostomy in the ambulatory patient and has fewer complications. It is accomplished by surgical incision on the left side of the neck under local anesthesia. A relatively large diameter rubber catheter may then be inserted. Subsequently, either a food formula or blenderized home food can be given in this way. As a note of caution: whenever general anesthesia becomes necessary avoid curare or succinylcholine which can be life-threatening in ALS patients. See *Table VIII*.

Dyspnea

Patients with ALS should be followed with pulmonary function tests, especially maximum expiratory pressure (MEP) and forced vital capacity (FVC). The effect of edrophonium (Tensilon) on these two variables should be assessed. If improvement is found, a daily trial of anticholinesterase medication (Mestinon) is advisable. Tracheostomy should be considered in several circumstances: when mechanisms protecting the larynx from aspiration are no longer efficient; when secretions are difficult to control; and when alveolar hypoventilation is established and derangement of blood gases develops. The earliest appearance of the latter occurs during sleep, the symptomatic presentation being restless sleep with multiple awakenings and drowsiness during day time. Thus, ALS patients have a form of sleep apnea, and sudden death has been seen on a number of occasions.

TABLE IX
TREATMENT OF DYSPNEA

Instruction in pulmonary toilet early in disease
Pneumovax and flu vaccine
Hospitalize and vigorously treat bronchitis or pneumonia promptly
Pyridostigmine (Mestinon 60 mg) may improve pulmonary function if complicated by myasthenic syndrome
Avoid aspiration — tracheostomy or surgical closure of larynx (eliminates remaining speech)
Mechanical ventilatory assistance after thorough discussion with patient and family

Patients with ALS should be treated vigorously when mild respiratory infections are detected and preventive measures, such as pneumococcal vaccine (Pneumovax), should be administered regularly.

A non-invasive mechanical ventilator (Pneumobelt) or cuirass respirator should be tried in every case requiring ventilatory assistance. The decision for permanent life support measures including mechanical ventilation should be made on an individual basis. In such instances, the decision is a group one involving not only the patient and physician, but the entire family as well.[6] See *Table IX*.

Conclusions

A brief review of the theories of etiopathogenesis in ALS, the major, sporadic form of adult MND has been presented. The theories, as developed from reviews of therapeutic trials during the last 40 years, have been compiled in attempting to arrive at a unifying hypothesis. Our hypothesis is that increased activity of extracellular proteases in muscle connective tissue and at the NMJ initiates and continues nerve-muscle degeneration. This hypothesis fits the "dying-back" model to explain motor neuron loss in ALS. A review of nerve-muscle adhesion, role of the BL and protease activity at the NMJ in intact nerves and after denervation has also been presented.

A rational approach to therapy, based on this theoretic presentation, using exogenous protease inhibitors has been described. Finally, symptomatic therapy, to demonstrate the willingness of the neurologic physician along with his or her colleagues to treat an "untreatable" degenerative neurological disease, has been reviewed.

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Acknowledgments

Studies included in this review were partially funded by grants from the Muscular Dystrophy Association, the ALS Society of America, and the Medical Research Service of the Veterans Administration. In addition, support by the Charles L. Betzelberger ALS Research Fund and the ALS Regional Research Center at KUMC are gratefully appreciated.

Laryngeal Carcinoma

Radiation Therapy for Early Cancers of the Larynx

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LARYNGEAL CARCINOMA accounts for approximately 29 per cent of all head and neck cancers.[1] The true vocal cords (TVC) are most commonly involved. Due to the relative lack of lymphatics in the true vocal cords the tumors are highly curable with local treatment such as surgery or radiation therapy. Lymph node metastasis occurs only when the tumor has invaded deeply into the underlying muscle or spreads beyond the glottic larynx.[2]

The anterior one-third of the TVC is the commonest site of origin. From there the tumor spreads to involve the anterior commissure and the opposite TVC. Extension to the supraglottic and subglottic areas also occurs. Involvement of the posterior-third of the TVC is rare. The majority of these tumors are well differentiated squamous cell carcinomas.

All patients should have a thorough work up which includes physical examination, routine blood chemistries, chest x-ray, panendoscopy, and biopsy of the laryngeal lesion. Once the diagnosis has been established the patient should be properly staged *(Table I)*. We will consider herein only the early lesions (T1N0M0).

Treatment Modality

Since both surgery and radiation therapy give excellent local control, the choice of treatment modality is influenced by the need to preserve a near normal voice. Radiotherapy has an advantage in that preservation of voice is possible. Stoicheff[3] examined the voice after radiation therapy in 227 patients with laryngeal carcinoma. Normal to near normal voice was reported in 83 per cent of patients. In a subsequent study an examination of the speaking fundamental frequencies in 22 patients with laryngeal cancer when compared to normal unirradiated patients revealed only slightly lower frequency (in Hz) in the irradiated group. Heavy smokers in both groups had a lower frequency indicating that smoking probably irritates the vocal cords. Similar results were reported by Karim[4] *et al.* and Colton.[5]

Radiation Technique

A wide variety of techniques are used in the treatment of laryngeal cancers.[6] Anatomically the TVCs are located 1 cm below the thyroid notch. A field between 5 x 5 cm to 6 x 6 cm centered 1 cm below the thyroid notch usually covers the treatment volume. Posteriorly the field should extend to the prevertebral fascia. Anteriorly there should be a fall-off beyond the skin. The most commonly used fields at UKSM are shown in *Figures 1* and *2*. In *Figure 1*, the open lateral fields result in a higher dose to the anterior commissure which may be well suited for patients in whom there is involvement of the anterior commissure. Use of 45 degree wedges as shown in *Figure 2* results in a reduction of the dose to the anterior commissure and produces a more even distribution of dose. If the posterior commissure is not involved, care should be taken to avoid irradiating the arytenoid cartilages for the entire treatment, especially if doses above 6000 rads are used. Failure to do so can result in edema of the arytenoid cartilage and persistent hoarseness of voice.

Radiation therapy is an effective treatment for early glottic cancers. There is a high control rate (85-90%) with preservation of the voice in a majority of patients. Surgical procedures can salvage 50-60 per cent of radiation failures and hence should be reserved for cases that fail primary radiation therapy.

Dose: A wide range of doses has been used. In general non-bulky lesions require 5500-6000 rads in 5.5 to 6.0 weeks while larger lesions require 6600 rads in 6.5 to 7.0 weeks.

Results: Local control in the range of 80-90 per cent can be achieved with radiation therapy *(Table II)*. Involvement of the anterior commissure does not portend an unfavorable prognosis as suggested by Kirchner[7] and Biller.[8] It has been our experience as well as others[9, 10] that these patients do as well as

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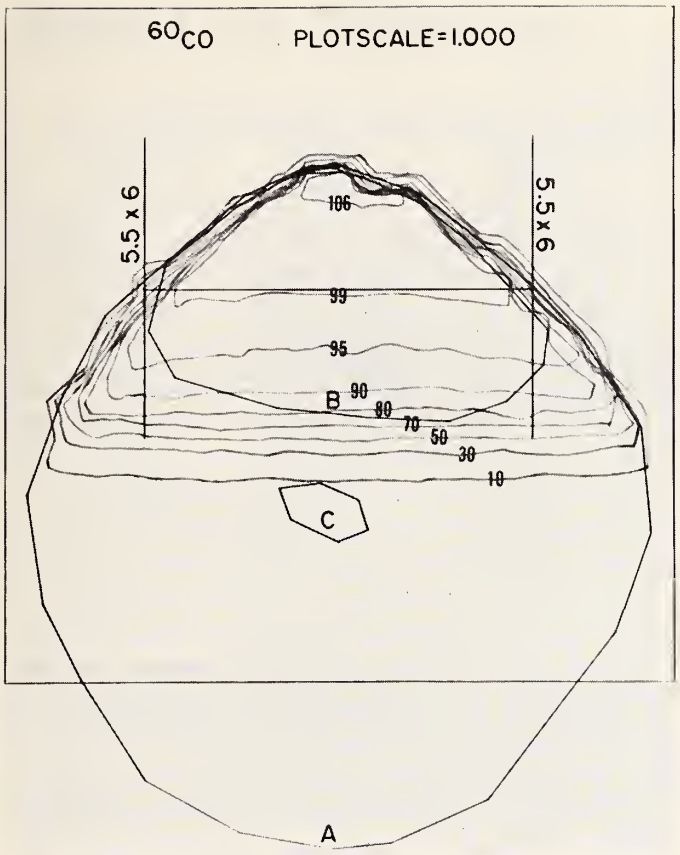


Figure 1. Isodose distribution using two open lateral fields. The "hot spot" is at the anterior commissure with rapid falloff posteriorly.

TABLE I
AJCC STAGING OF GLOTTIC CANCER

Primary Tumor (T)	
TIS	Carcinoma in situ
T1	Tumor confined to vocal cord(s) with normal mobility (includes involvement of anterior or posterior commissures)
T2	Supraglottic and/or subglottic extension of tumor with normal or impaired cord mobility
T3	Tumor confined to the larynx with cord fixation
T4	Massive tumor with thyroid cartilage destruction and/or extension beyond the confines of the larynx
Nodes (N)	
Nx	Nodes cannot be assessed
N0	No clinical positive node
N1	Single clinically positive homolateral node 3 cm or less in diameter
N2	Single clinically positive homolateral node more than 3 cm but not more than 6 cm in diameter or multiple clinically positive homolateral nodes none more than 6 cm
N3	Massive homolateral node(s), bilateral nodes, or contralateral node(s)
N3a	Clinically positive homolateral node(s), one more than 6 cm in diameter
N3b	Bilateral clinically positive nodes (in this situation, each side of the neck should be staged separately; that is, N3b: right, N2a; left, N1)
N3c	Contralateral clinically positive node(s) only
Distant Metastasis (M)	
MX	Not assessed
M0	No (known) distant metastasis
M1	Distant metastasis present
Stage Grouping	
Stage I	T1 N0 M0
Stage II	T2 N0 M0
Stage III	T3 N0 M0
Stage IV	T4 N0 or N1 M0 Any T N2 or N3 M0 Any T Any N M1

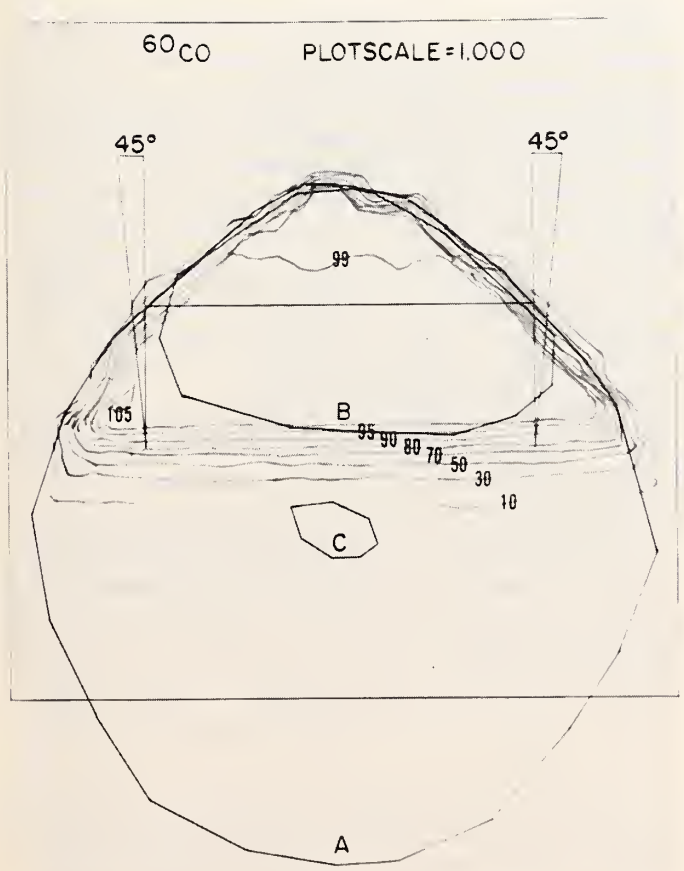


Figure 2. Isodose distribution using wedged lateral fields. Note there is a more homogenous dose distribution throughout the treatment volume.

TABLE II
RESULTS OF TREATMENT

	Total No. of Patients	*Radiation Failures*		*Surgical Salvage*		*Total Failure*		*Overall Control*
		#	%	#	%	#	%	
*Fletcher[11]	332	37	11.1	31/36	86.0	6/332	1.8	98.2
†Harwood[15]	383	54	14.0	34/54	62.9	20/383	5.2	94.8
Stewart[16]	168	12	7.1	10/12	83.0	2/168	1.1	98.9
Hawkins[17]	398	62	16.0	37/62	59.7	25/398	6.3	93.7
‡Hendrickson[18]	169	17	10.0	10/13	76.9	7/169	4.1	95.9
	1450	182	11.6	122/177	68.9	60/1450	4.1	95.9

* One patient did not undergo surgery.
† Includes 47 T1S stage patients.
‡ Four patients did not undergo surgery.

those without anterior commissure involvement.

Failure: The most common causes for failure are:

- Improper techniques that do not cover the tumor adequately.
- Undiagnosed extensions beyond the glottic larynx.
- In about 10 per cent of the cases no obvious explanation can be given. Fletcher[11] suggests that it may be that the plateau of the sigmoid curve for cure has been reached.

Salvage Surgery: Patients failing radiotherapy can be salvaged by hemi- or total laryngectomy.[12-14] *Table II* shows that a large percentage of patients can be salvaged by surgery resulting in a high degree of overall local control. The appropriate treatment for early glottic cancers therefore appears to be radiation therapy with surgical procedures reserved for patients who fail radiation therapy.

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Beta Cell Function

Restoration and Preservation of B-cells in Children With Type I Diabetes

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THE EXOGENOUS insulin requirement of most children with recent onset of diabetes decreases rapidly and predictably to a relatively low level (0.15-0.40 U/kg/day) when prompt insulin treatment is given.[1-3] This state of partial remission, except in rare cases, occurs but once and persists for a variable period of time. In 1971,[4] we reviewed the records of 63 children with recent onset of diabetes to compare responses of children with early diagnosis and prompt insulin replacement with the responses of children with delayed diagnosis and treatment. We found that the earlier the diagnosis was made and insulin was given, the lower the maintenance insulin requirement and the easier it was to attain and maintain a high degree of metabolic control with minimal risk of hypoglycemia. When parents continued the treatment plan at home, partial remission persisted for as long as four to five years. In 1978, we reviewed the records of an additional 90 children under continuous care in our clinic since onset of diabetes who had remained in a high degree of control. After stabilization, the insulin requirement of these children 7-12 years of age was 0.16-0.42 (mean 0.32) U/kg/day. Forty-four remained in partial remission (*i.e.* required less than 0.60 U/kg/day to maintain aglycosuria and to permit normal growth) for 4-60 (mean 26) months; 40 at the time of their last observations were still in partial remission for 8-42 months; and the other six were older children in their growth spurt who required from 0.64 to 0.68 U/kg/day after stabilization. We also have observed that the maintenance insulin requirements for adolescents who have completed their growth spurt before the onset of diabetes is lower and extends for longer periods of time than for younger children as reported by Madsbad *et al.*[5]

The need for more objective criteria to classify degrees of metabolic control in patients with diabetes has been recognized for many years. Recent studies have demonstrated the usefulness of glycohemoglobin (HbA_{1c}) levels to evaluate more objectively the degree of hyperglycemia in patients with insulin dependent and those with maturity onset diabetes.[6, 7] We have refined and published the criteria used in our diabetic clinic to classify varying degrees of metabolic control; a statistically significant correlation was found between the clinic control ratings and the HbA_{1c} levels ($p < 0.01$ between varying control groups).[8]

Pancreatic beta cell function was assessed by determination of C-peptide levels in ten normal subjects and 60 children and young adults with insulin-dependent diabetes. This study provides additional evidence that early and adequate insulin replacement is important to restore beta cell function, and that maintaining a high degree of control conserves beta cell function.

A highly sensitive radioimmunoassay recently has become available for the determination of human C-peptide reactivity.[9] C-peptide is cleaved from proinsulin during its conversion to insulin, and is secreted by the beta cell in equimolar amounts with insulin. C-peptide provides an accurate index of endogenous insulin secretion even in the presence of exogenous insulin. The availability of this assay prompted us to investigate the endogenous secretion of insulin in diabetic children with varying exogenous insulin requirements, maintained in "excellent to good" control, during the period of partial remission. Selected children with longer duration of diabetes in both higher and lower degrees of control also were studied.

Materials and Methods

Patients. The study population is composed of 60 children with overt insulin-dependent diabetes, and ten healthy children and young adults with no family history of diabetes and normal oral glucose tolerance tests who served as controls. There were 34 diabetic boys and 26 diabetic girls varying in age of onset

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from 4.50 to 15.25 years (mean 10.5) and 7.0 to 13.5 years (mean 10.5), respectively.

Research groups consisted of normal subjects and patients with insulin-dependent diabetes as follows:

- Nine girls and six boys with recent onset of diabetes, studied within one month after nutritional repletion and establishment of insulin requirement, and at periodic intervals during the following six months.
- Nine girls and 24 boys who attained and maintained "excellent to good" control documented by serial HbA_{1c} levels and diurnal blood glucose levels; and three boys and three girls known to have been in lower degrees of control for extended periods of time.
- Two girls and two boys who came under care in our clinic after receiving conventional treatment elsewhere — *i.e.*, one boy and one girl who had remained in fair control since onset of disease; and one boy and one girl who had remained in "fair to poor" control since onset of disease.
- Five girls and one boy who were above expected weight for height at time of diagnosis. Two of the girls were obese, *i.e.*, weight was greater than two standard deviations (SD) above average weight for actual height.

The total number of diabetic children in different subgroups is 64 because four of the subjects are included in two groups.

All of the diabetic children had classical symptoms of diabetes two to four weeks prior to diagnosis and admission to our medical center. Each of them had gross glycosuria and confirmed fasting and postprandial hyperglycemia. Twenty-four were ketoacidotic at the time of admission with weight loss ranging from 9-20 per cent. All of the six overweight children were ketotic but not acidotic.

Methods. Informed consent was obtained from the parents and also from the older children prior to their participation in the study.

To assess pancreatic beta cell function, fasting and one and two hour postprandial serum glucose and C-peptide levels were determined in the normal and diabetic subjects. Serum insulin also was determined in the normal subjects. Testing was done after a ten hour overnight fast and after the ingestion, within about five minutes, of 20 per cent of each subject's estimated daily caloric requirement based on age and weight, as a standard liquid breakfast (Sustacal — Mead Johnson) containing 25 per cent protein, 55 per cent carbohydrate, and 20 per cent fat. None of the diabetic subjects received insulin on the morning of the test.

Serum insulin determinations were done in triplicate by a modification of the double antibody method of Morgan and Lazarow.[10] The sensitivity of our assay allowed for the detection of 5 μU/ml of insulin and a reproducibility within an assay of 3.6 per cent coefficient variation (CV) and between assays of 7.9 per cent CV.

Serum glucose determinations were made on Somogyi filtrate by the O-Toluidine method using a Technicon autoanalyzer. The variability in the serum glucose was within 3 mg/dl.

C-peptide determinations were done by radioimmunoassay in the laboratory of Dr. Arthur Rubenstein[11-13] by use of an antiserum supplied by Binder and Faber. The sensitivity of the assay was 0.10 ng/ml and the cross reactivity with proinsulin was less than 10 per cent.[9]

Glycolated A_{1c} hemoglobin determinations were done by the column chromatography method of Trivelli[14] modified by the use of a Technicon peristaltic pump in order to apply moderate pressure to the column flow rates. All specimens were analyzed in our research laboratories by the same experienced technician within a few hours after collection or frozen at −70C and then analyzed a few days later. Mean ± 1 SD A_{1c} values in our laboratory are: Normal subjects 4.26 ± 0.49; diabetic children in "excellent" control 4.99 ± 0.70; in "good" control 6.36 ± 0.50; in "fair" control 8.69 ± 0.68; and in "fair to poor" control 10.84 ± 0.87. Thirty-two of the diabetic subjects classified to be in "excellent to good" control also had diurnal blood sugar determinations done under controlled conditions in our Clinic Research Center. Their mean ± 1 SD glucose values mg/100 ml were: 6 hour (h), 81 ± 13; 9 h, 96 ± 36; 12 h, 79 ± 16; 14 h, 126 ± 44; 16 h, 104 ± 28; 20 h, 76 ± 26; 22 h, 97 ± 22; 3 h, 77 ± 16; and 6 h, 88 ± 18.

Results

The clinical and laboratory data of individual and homogenous groups of diabetic subjects were critically reviewed, tabulated, and analyzed. Ten normal non-obese children and adolescents (five girls and five boys) had a standardized Sustacal tolerance test as a control group. All of these control subjects had previously had normal serum glucose and insulin concentrations during oral glucose tolerance tests with values between the 84th and 16th percentiles.[15]

The mean C-peptide, insulin, and glucose values of these ten healthy control subjects are depicted in *Figure 1* and the mean ± 1 SD values also are recorded in the caption. The closely similar curves for C-peptide and insulin with a very narrow excur-

TABLE I
RESULTS OF STUDIES

Group A — Studies done within one month after nutritional repletion and when maintenance insulin requirement was determined.

Group B — Studies done one to six months after attaining and maintaining a high degree of control.

Subject	Age at onset yrs	Sex	Wks*	Insulin U/kg	HbA_{1c}	C-peptide / glucose Fasting	C-peptide / glucose 1 hr	C-peptide / glucose 2 hr	Wks*	Insulin U/kg	HbA_{1c}	C-peptide / glucose Fasting	C-peptide / glucose 1 hr	C-peptide / glucose 2 hr
1 R.D.	11	F	2	0.39	8.3	1.75 / 110	3.18 / 194	4.54 / 200						
2 R.S.	11	M	2	0.34	10.3	1.46 / 95	1.72 / 212	2.18 / 221						
3 R.C.	11½	M	3	0.22	8.3	0.60 / 74	1.50 / 182	3.00 / 132						
4 P.H.	12	F	3	0.24	12.4	0.50 / 48	2.80 / 121	4.70 / 113	22	0.30	7.5	2.71 / 163	4.36 / 346	4.52 / 387
5 D.H.	10	F	3.5	0.25	8.2	0.89 / 85	2.63 / 193	3.74 / 155	8	0.24	6.3	1.35 / 84	3.41 / 151	3.64 / 113
6 D.M.	7	M	4	0.30	12.0	0.65 / 106	2.40 / 211	2.10 / 182	12	0.30	7.4	1.00 / 111	4.00 / 172	3.90 / 193
7 A.P.	11	F	4	0.50	10.4	0.44 / 123	1.40 / 213	1.80 / 229	12	0.70	6.5	0.97 / 98	2.80 / 203	2.50 / 231
8 S.R.	10	F	4	0.36	8.6	0.56 / 98	2.70 / 127	2.60 / 92	24	0.41	6.0	2.52 / 129	4.36 / 184	5.20 / 227
9 M.S.	9	M	4	0.26	9.1	0.55 / 125	1.10 / 207	1.50 / 267	16	0.28	5.4	1.30 / 91	3.90 / 208	3.10 / 149
10 J.S.	11	M	4	0.26	8.4	0.26 / 52	1.68 / 174	1.58 / 159	8	0.26	5.3	0.82 / 86	3.25 / 115	3.15 / 103
11 J.W.	11	F	4	0.25	8.2	0.03 / 86	1.60 / 150	2.43 / 85	12	0.26	4.6	1.90 / 99	2.60 / 186	2.30 / 236
12 S.E.	9	F							5	0.33	7.2	1.31 / 88	3.29 / 199	3.85 / 171
13 K.C.	9	F							8	0.34	6.4	1.74 / 68	2.35 / 198	3.60 / 110
14 M.S.	16	M							10	0.28	6.5	1.91 / 81	2.97 / 111	3.95 / 86
15 S.P.	9	F							16	0.30	5.3	1.10 / 86	3.10 / 158	3.20 / 131
C-peptide	mean ±1 SD					0.70 ±0.47	2.07 ±0.66	2.74 ±1.08	C-peptide	mean ±1 SD		1.55 ±0.59	3.37 ±0.63	3.58 ±0.78
glucose	mean ±1 SD					91 ±24	180 ±32	167 ±56	glucose	mean ±1 SD		99 ±24	186 ±57	178 ±80

* After treatment.

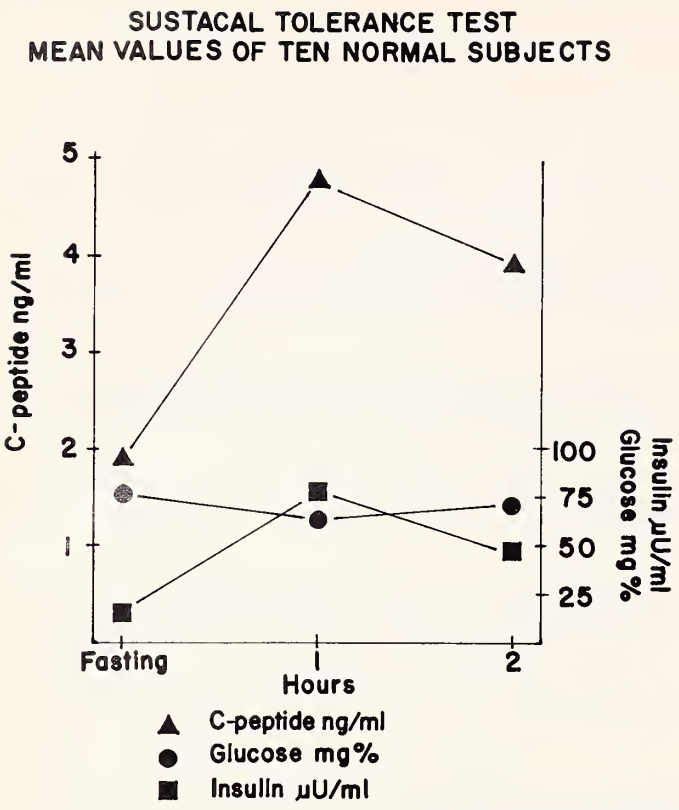


Figure 1. Mean fasting, one and two hour C-peptide, insulin and glucose values of ten healthy control subjects with no family history of diabetes and with normal oral glucose tolerance tests. The mean ± 1 SD fasting, one and two hour C-peptide values (ng/ml) were: 1.95 ± 0.8, 4.8 ± 1.6 and 3.0 ± 1.6.

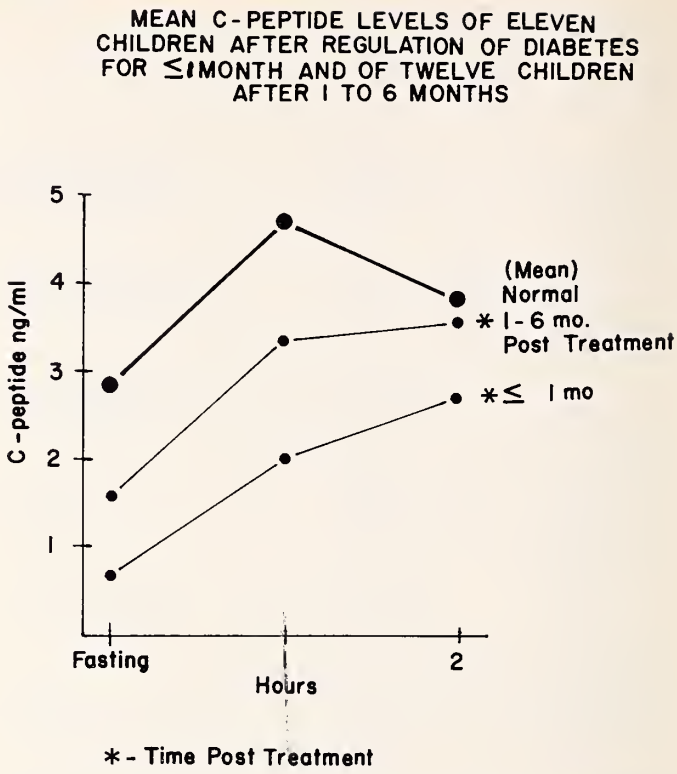


Figure 2. Mean fasting, one and two hour postprandial C-peptide values of children with recent onset after regulation of diabetes, for less than one month and after one to six months.

sion of blood sugar values are demonstrated.

Table I records and *Figure 2* depicts the fasting, one hour and two hour postprandial C-peptide and blood glucose values of two groups of children with recent onset of insulin-dependent diabetes, after receiving a standard liquid breakfast. Group A *(Table I)* are subjects arranged in order of increasing time from diagnosis who were studied within one month after treatment began, when they had attained nutritional repletion and a stable insulin requirement. Subjects 1 and 2, with higher fasting C-peptide levels, had minimal weight loss at the time treatment began. Within the first week after treatment, the HbA_{1c} values of the 15 subjects in Group A were elevated, ranging from 13.8 to 9.6 per cent. At the time of the study, as indicated in *Table I*, the HbA_{1c} values of 11 subjects had decreased to 12.4 to 8.2 per cent but were still well above the normal range. Group B are subjects maintained in metabolic control who were studied between one and six months after treatment began. Group B included eight repeat tests of subjects 4 through 11 included in Group A. Note the consistent improvement not only in the higher fasting C-peptide levels but also in the greater C-peptide response after the ingestion of a standard meal in the Group B subjects who had the test repeated after attaining and maintaining "good to excellent" metabolic control, as documented by HbA_{1c} values which had decreased to only slightly above the normal range. The increase in C-peptide values of the eight subjects common to both groups is significant at fasting ($P < 0.01$) and one hour ($P < 0.01$). At the two hour time period $P = 0.06$. Note that the mean fasting glucose levels 91 *vs* 99 mg/100 ml in each group are within the high normal range, whereas the postprandial glucose values are, with only a few exceptions, grossly elevated, confirming the insulin dependency of these subjects, none of whom received insulin replacement on the day of the test.

Table II summarizes the clinical and laboratory data of all of the diabetic subjects who remained in metabolic control. The subjects are subdivided on the basis of their daily insulin requirement expressed as U/kg/day. *Figure 3* demonstrates the consistent progressive decrease in C-peptide levels as the ex-

TABLE II
CLINICAL AND LABORATORY DATA OF DIABETIC CHILDREN MAINTAINED IN HIGH DEGREE OF METABOLIC CONTROL SUBDIVIDED ON BASIS OF EXOGENOUS INSULIN REQUIREMENT.

Insulin requirement U/kg/day	<0.30		0.30-0.40		0.40-0.50		0.50-0.60		0.60-0.70		0.70-0.80		>0.80	
No. of Subjects	18		17		11		10		10		11		12	
	*	†	*	†	*	†	*	†	*	†	*	†	*	†
Age at onset yrs	10.7	7/16	11.0	9/16	11.0	9/12	10.0	5/15	9.0	7/15	9.9	4/15	10.8	7/14
Duration yrs	0.9	0.1/2.1	0.7	0.1/4.3	1.0	0.1/2.3	1.6	0.9/3.8	2.4	1.0/4.3	2.4	0.9/4.6	3.8	0.5/4.6
Insulin/kg/day	0.21	0.10/0.29	0.34	0.34/0.39	0.44	0.40/0.47	0.57	0.50/0.59	0.65	0.62/0.68	0.75	0.70/0.79	0.94	0.84/1.14
% HBA_{1c}	5.6	4.2/7.1	6.7	4.6/8.6	6.9	4.5/8.5	6.4	5.2/7.3	5.8	4.8/7.2	6.7	5.2/8.6	7.4	6.4/8.6
Fasting C-peptide	1.70±0.78		1.54±0.64		1.39±0.53		1.33±0.40		1.08±0.23		0.80±0.41		0.65±0.33	
1 hr C-peptide	3.63±1.15		3.16±1.16		2.50±0.90		1.89±0.71		1.91±0.86		1.23±0.62		0.96±0.42	
2 hr C-peptide	3.43±1.09		3.35±1.06		2.57±1.04		2.37±0.79		1.82±0.60		1.32±0.68		0.83±0.45	

* Mean
† Range
± 1 SD

ogenous insulin requirement increases. The Kruskal-Wallis test was used to determine whether the seven groups depicted in *Figure 3* were different relative to the C-peptide levels. The P value for each of the time periods was less than 0.001. Regression lines were computed considering C-peptide as a function of the exogenous insulin requirement for each of the three time periods. The slope of each line was negative and was significantly different from zero. The P value was less than 0.001 in all time periods.

A Sustacal tolerance test was done in an 8-year-old diabetic girl before she received insulin replacement therapy. Three weeks before admission to our clinic this child, during an upper respiratory infection, was seen by her family physician. At this time she was found to have glycosuria with a fasting blood glucose value of 134 mg/100 ml and a two hour postprandial value of 245 mg/100 ml. Although her symptoms diminished and her urine became sugar free within a few days, she was referred to our clinic for evaluation and management. At the time of admission to the Medical Center she had glycosuria of 1 per cent during a standard oral glucose tolerance test with a high normal fasting blood glucose level but the one-half, one, and two hour glucose values all were more than 2 SDs above mean values for normal girls. Her serum insulin levels also were more than 1 SD below mean values for normal

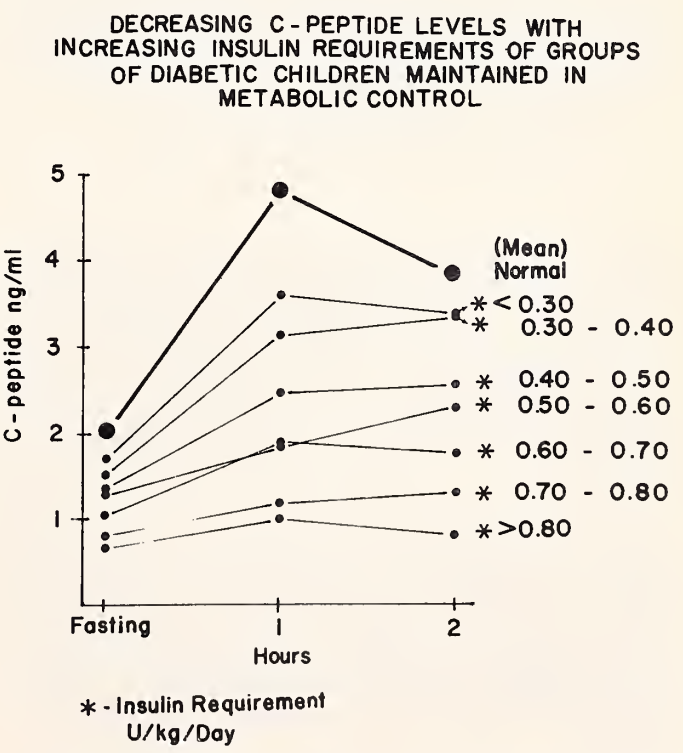


Figure 3. Consistent, progressive decrease in fasting and postprandial C-peptide levels with increasing exogenous insulin requirement in groups of well controlled diabetic children. The rate of C-peptide release is also decreased as the insulin requirement increased.

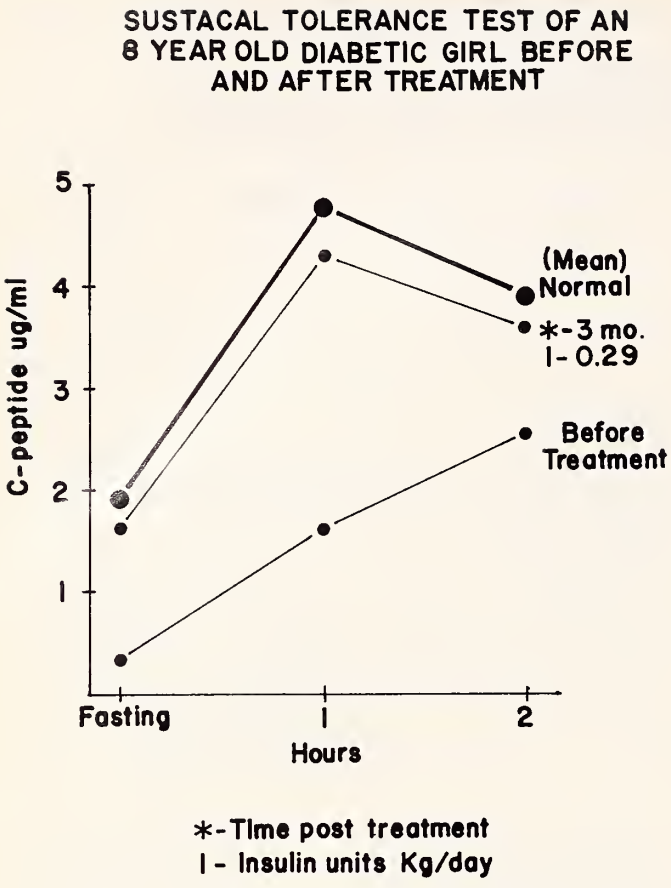


Figure 4. Note the low fasting C-peptide value and delayed response to the ingestion of food at the time of diagnosis, and the approximately normal response after maintaining metabolic control for three months reflecting restoration of B-cell function.

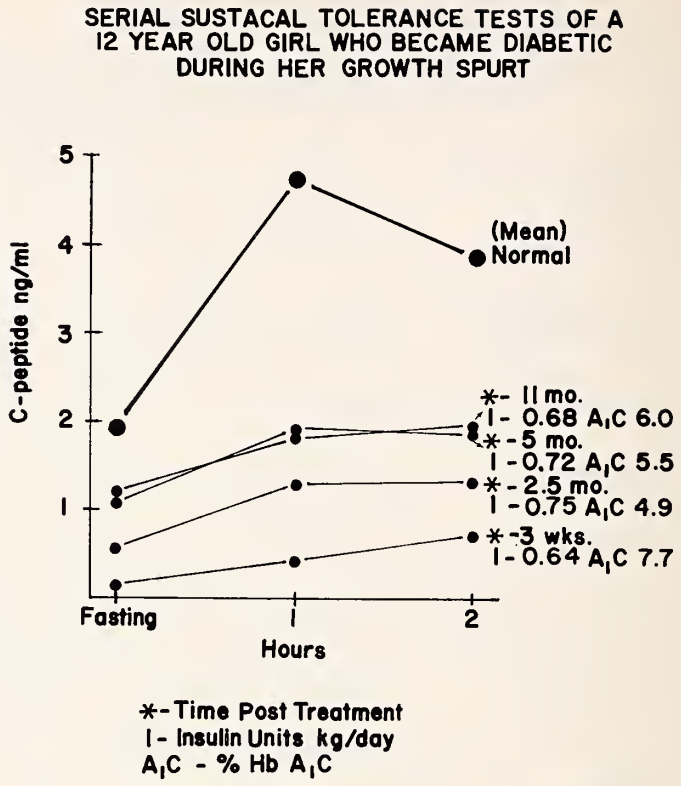


Figure 5. Serial Sustacal tolerance tests of an adolescent girl during her prepubertal growth spurt. Note the progressive restoration and preservation of B-cell function after the period of accelerated growth.

girls.[15] A Sustacal tolerance test was done the day after the oral glucose tolerance test and just prior to the initiation of insulin replacement therapy. The test was repeated three months later after she had remained in metabolic control with an insulin requirement of less than 0.3 U/kg/day. Results of the tests done before and after treatment are depicted in *Figure 4*.

Figure 5 illustrates the loss of beta cell function at onset of the disease in a prepubescent diabetic girl and the subsequent partial restoration of function after maintaining metabolic control. *Figure 6* contrasts the degree of this loss and restoration of function with the same process in a postpubescent diabetic girl.

Four diabetic children included in the study had received conventional treatment for 8-22 months before admission to our clinic. On the basis of history and clinical condition at the time of admission two were classified as having been in "fair to good" control and the other two in "fair to poor" control. Before admission to our clinic the children in Group A had received one or two daily insulin injections and structured meals. Data from their referring physicians and from their home records indicated only minimal and transient glycosuria after diagnosis and insulin replacement. Both subjects in Group A were in a good nutritional state at the time of admission to our clinic. In contrast, children in Group B had continued to have varying degrees of glycosuria most if not all of the time since diagnosis and had histories of moderately severe hypoglycemic episodes requiring emergency medical treatment. Both subjects in Group B had accelerated linear growth and an improved nutritional state after maintaining metabolic control. Sustacal tolerance tests were done one to three months after the subjects had attained metabolic control and their insulin requirements were determined. Results of the tests are depicted in *Figure 7*.

Table III records the clinical and laboratory data of five girls (four postpubescent) and one school age boy who were at least 1 to 2 SDS above average

TABLE III
C-PEPTIDE RESPONSES OF SIX DIABETIC SUBJECTS WHO WERE OVERWEIGHT FOR HEIGHT AT THE TIME OF ONSET OF OVERT DIABETES

Subject	Sex	Age at Onset Yr Mo	Duration Yr Mo	Insulin U/kg	HbA_{1c}	C-Peptide Fasting	C-Peptide 1 hr	C-Peptide 2 hr
1 K.S.*	F	12-6	1-5	0.15	8.2	10.50	11.30	13.10
			1-10	0.15	7.3	8.90	10.10	11.30
			2-0	0.14	8.7	4.00	5.17	5.59
			2-10	±0.40	8.1	4.32	5.81	5.98
2 BM.*	F	8-1	0-6	<0.10	8.6	2.69	3.94	3.42
3 P.H.	F	12-6	0-6	0.30	8.5	2.71	4.36	4.52
4 T.T.	M	12-2	0-11	0.11	4.9	2.40	4.60	4.50
			2-0	0.17	6.2	2.76	6.80	4.25
5 P.C.	F	12-2	2-6	0.10	4.2	1.60	3.90	4.40
6 P.P.†	F	13-6	3-4	±0.40	11.4	0.75	0.71	1.03

* Subjects 1 and 2 were obese, *i.e.* weight >2 SD above average weight for actual height.
† Subject 6 was known to have been in "fair to poor" control at time of test and for more than one year before the test.

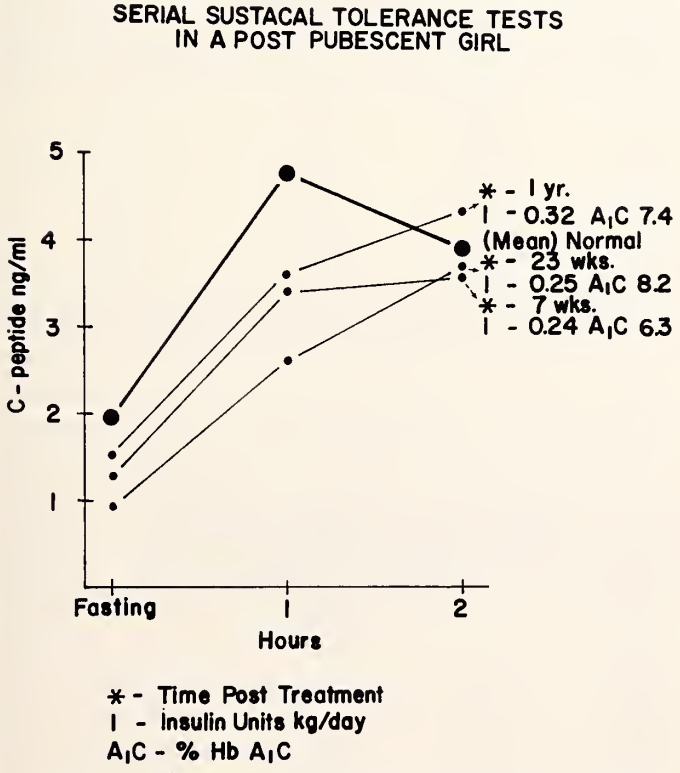


Figure 6. Restoration and preservation of B-cell function in a postpubescent diabetic girl. Note that her fasting C-peptide levels are only modestly decreased and her C-peptide response to the ingestion of food is relatively satisfactory.

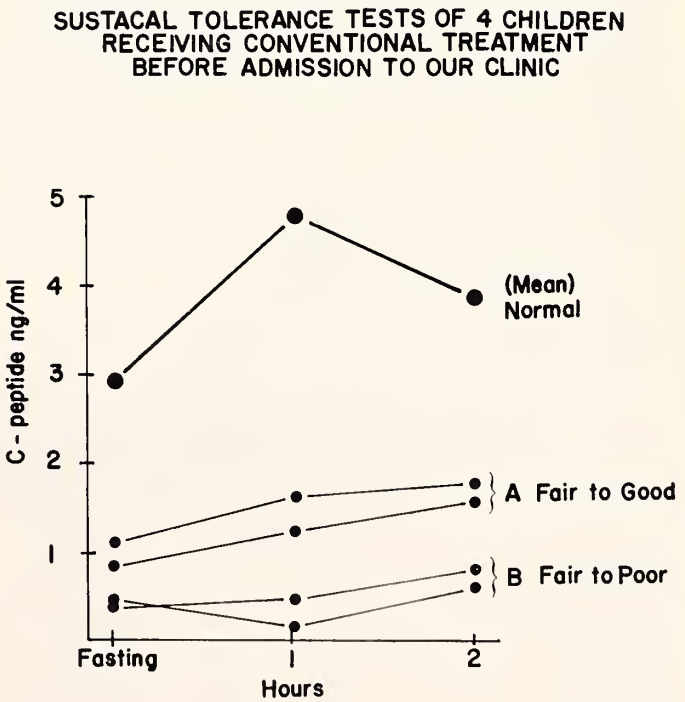


Figure 7. Response of two children (Group A) maintained in "fair to good" control compared to two children (Group B) maintained in "fair to poor" control for extended periods of time before admission to our clinic.

TABLE IV

SERIAL OBSERVATIONS OF FOUR ADOLESCENT INSULIN-DEPENDENT DIABETIC SUBJECTS (2 BOYS AND 2 GIRLS) WHO WERE UNABLE TO MAINTAIN A HIGH DEGREE OF CONTROL DURING THEIR PREPUBESCENT GROWTH SPURT CONCURRENT WITH THEIR RAPIDLY INCREASING INSULIN REQUIREMENTS

Subject	*Age at Onset*	*Duration* Yr	Mo	*Insulin U/kg*	HbA_{1c}	*C-Peptide* Fasting	1 hr	2 hr
1 R.W.	10-6	0	11	0.45		1.62	3.15	2.41
		1	3	0.57		1.26	2.40	2.68
		2	3	±1.00	8.6	0.06	0.64	0.24
2 C.H.	11-2	1	0	0.62	5.6	1.19	2.40	1.18
		2	6	±0.84	8.1	0.91	1.20	0.71
3 P.F.	11-3	1	0	±0.76	8.4	1.23	1.86	1.23
		1	2	±0.76		0.96	1.30	1.26
		2	6	±0.84	12.3	0.36	0.26	0.14
4 M.M.*	10-2	0	6	±0.86	7	0.90	0.98	1.06
		0	10	±0.90	8.3	0.72	0.86	0.48

* Subject 4 had been symptomatic for four weeks and at time of admission to the hospital was in severe ketoacidosis with a weight loss exceeding 10 per cent of her body weight.

TABLE V

OBSERVATIONS OF SEVEN SUBJECTS WITH DIABETES FOR MORE THAN ONE YEAR IN LOWER DEGREES OF CONTROL DOCUMENTED BY ELEVATED HbA_{1c} LEVELS

Subject	*Duration* Yr	Mo	*U/kg**	HbA_{1c}	*C-Peptide* Fasting	1 hr	2 hr
P.F.	2	6	0.84	12.3	0.36	0.26	0.14
C.H.	2	6	0.84	8.9	0.91	1.20	0.71
J.N.	2	1	0.60	12.4	0.42	0.13	0.35
M.S.	1	1	0.70	8.6	0.40	1.07	1.24
R.W.	2	3	1.00	8.6	0.06	0.64	0.24
M.P.	1	3	0.60	8.9	1.49	1.10	1.50
P.P.	3	0	0.62	11.4	0.75	0.71	1.03

* Insulin is dosage, not requirement.

TABLE VI

OBSERVATION OF SIX SUBJECTS WITH HIGH INSULIN REQUIREMENTS BUT WHO MAINTAINED GOOD METABOLIC CONTROL (CONFIRMED BY HbA_{1c}) DESPITE LOW C-PEPTIDE LEVELS INDICATING MINIMAL OR ABSENT RESIDUAL BETA CELL FUNCTION

Subject	*Duration* Yr	Mo	*Insulin U/kg**	HbA_{1c}	*C-Peptide* Fasting	1 hr	2 hr
C.H.	2	5	1.14	6.0	0.24	0.50	0.52
D.G.	3	4	0.78	6.9	0.48	0.43	0.38
D.G.	3	6	0.77	7.5	0.52	0.25	0.18
B.H.	4	6	0.70	7.2	0.27	0.25	0.18
J.H.	1	7	1.14	6.4	0.68	1.14	1.22
J.S.	0	8	0.70	5.9	0.45	0.19	0.60
B.S.	4	4	0.62	7.2	0.59	1.40	1.80

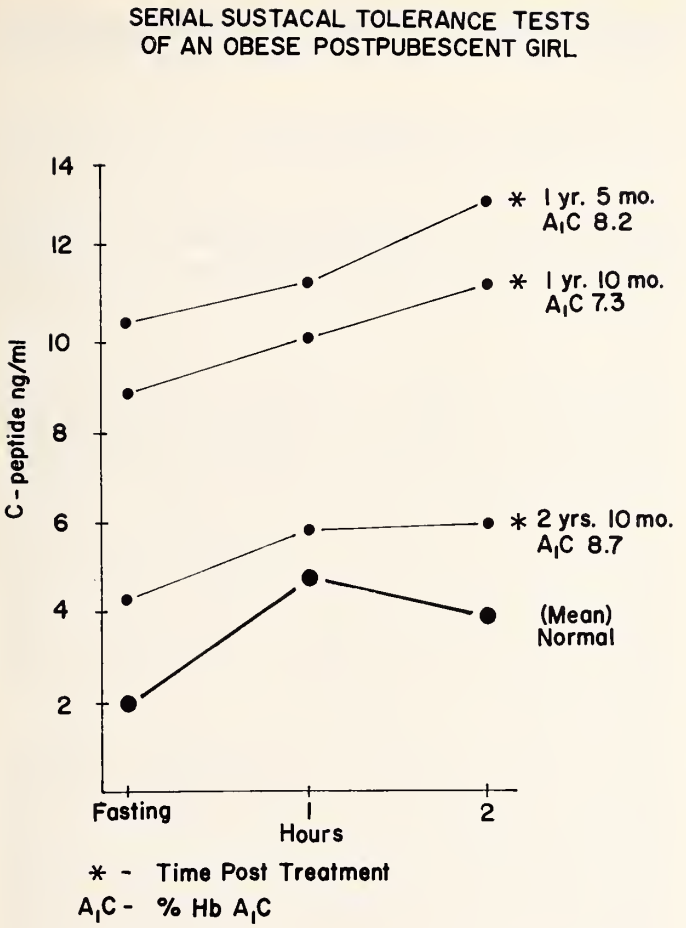


Figure 8. Elevated but delayed rate of secretion of endogenous insulin in a 13-year-old obese postpubescent girl with progressive decrease in C-peptide levels with time.

weight for height before the onset of overt diabetes. All were ketotic but not ketoacidotic at the time of diagnosis with gross glycosuria and repeated blood glucose values >200 mg/100 ml. Subjects 1 and 2 were obese, *i.e.* their weights were >2 SDs above average weight for actual height. (*Figure 8* demonstrates the elevated value and delayed rate of endogenous insulin secretion in subject 1 K.S., a 13-year-old postpubescent girl.) Subject 2 B.M. (the younger obese school girl), was developmentally retarded. The oldest postpubescent girl, subject 6 P.P., remained in good control during a two year period with nutritional guidance. For 16 months before the Sustacal tolerance test she failed to adhere to her meal plan, took varying doses of insulin erratically, and regained excessive weight. Note that her HbA_{1c} value was 11.4 per cent indicating "fair to poor" metabolic control; C-peptide values demonstrate only minimal residual beta cell function.

Table IV demonstrates the loss of beta cell function in four patients who were in a lower degree of metabolic control during adolescence. *Table V* demonstrates loss of beta cell function in seven other subjects of varying ages who were unable to maintain "good" control.

Table VI demonstrates the success some juvenile diabetics have had in maintaining a high degree of metabolic control (confirmed by HbA_{1c} levels) despite high exogenous insulin requirements and loss of endogenous insulin secretion.

At the end of the three year study period, 47 of the 60 insulin dependent diabetic subjects had fasting C-peptide values <0.8 ng/ml as well as one and two hour postprandial values >1.20 ng/ml, indicating some endogenous insulin secretion. Nine had C-peptide values <0.5 ng/ml at each time period, and four had even lower values indicating minimal and questionable residual beta cell function.

Discussion

Insulin deficiency is the basic underlying defect in children with Type I (juvenile) diabetes. In the past it has generally been assumed that most diabetic children have minimal residual beta cell function at the time of onset of their disease and the ability to secrete endogenous insulin persists for only a short period of time.[3] The recent development of a more sensitive laboratory method to measure C-peptide levels (as low as 0.10 ng/ml) has made it possible to observe the beta cell secretory capacity of the pancreas during the course of insulin-dependent diabetes.

Rubenstein and Gonen[16] and Gonen *et al.*[17] have reviewed the literature and published C-peptide studies of children and young adults with insulin-dependent diabetes. Assessment of pancreatic insulin reserve has been done by measuring C-peptide levels after stimulation by either the ingestion of a meal or intravenous injection of 1 mg of glucagon. Hendricksen, Faber, and Binder[18] found an increase above fasting C-peptide levels in response to both of these challenges in 64 insulin-dependent diabetic subjects, and a significant correlation between the magnitude of the response and the fasting C-peptide level. Our method of assessing beta cell function is similar to that used by them and by Ludvigsson[3] and the responses of both our normal and diabetic subjects to a comparable challenge closely resemble the responses observed by these investigators. Faber and Binder[19] also found that the diabetic subjects with the highest fasting C-peptide concentrations had the lowest exogenous insulin requirements and a more stable metabolic status. Ludvigsson and Heding[3] did

serial fasting C-peptide determinations on 12 diabetic children for about one year after the time of diagnosis. At the time of diagnosis all subjects had very low but measurable fasting C-peptide values. At about one month after insulin replacement therapy, all of them had higher fasting C-peptide values. During the subsequent nine months with conventional management there was a steady decrease in C-peptide levels with values below the normal range except in one of the children. These authors also did fasting C-peptide determinations on 96 diabetic children who were treated in their clinic for more than two years. Treatment according to them consisted of a single daily dose of an intermediate acting insulin, a well balanced diet, and regular physical exercise. When the single daily injection of insulin failed to maintain satisfactory control, the insulin therapy was changed to twice daily injections with the addition of a short acting insulin. At the time of the study, 55 children were receiving a single daily insulin injection, and 41 were receiving twice daily injections. Thirty-five per cent of their patients had detectable fasting C-peptide levels (values >0.11 ng/ml). An index of metabolic control was calculated from home records of fractional urinary glucose analysis. A significant correlation was found between C-peptide levels and the diabetic control index in the children in whom more intense treatment was given at time of onset of diabetes.

Gonen *et al.*,[17] using HbA_{1c} levels, have confirmed these observations. They found that nonobese insulin-dependent diabetic subjects with fasting C-peptide concentrations above 0.1 pmol/ml (0.29 ng/ml) had significantly better metabolic control than did those with lower values. They also found that patients with onset after 20 years of age had higher fasting C-peptide values and higher values after stimulation. They postulated that good metabolic control may contribute to the preservation of insulin secretory capacity and concluded that additional studies are needed to resolve this important question. Mirouze *et al.*,[19] by means of an external artificial pancreas, also have recently demonstrated that the institution of physiologic control shortly after the diagnosis of diabetes is associated with enhanced preservation of beta cell function. During the period of partial remission we have not found it necessary to use insulin infusion pumps to attain and maintain essential physiologic control.

For children with insulin-dependent diabetes, we define insulin requirement to be that amount of exogenous insulin per unit of body weight required daily to maintain aglycosuria and approximately normal blood glucose sugar levels when the child is ingesting adequate calories and essential nutrients to permit normal growth and avoid excessive weight gain.[20, 21]

By extensive clinical experience we have found that if the diabetic child receives early and adequate insulin replacement therapy, the exogenous insulin requirement after nutritional repletion usually will remain relatively constant from day to day. We also have found that the rate of increasing insulin requirement is usually predictable and intimately interrelated with the normal increase in body weight for height and the normal growth and maturation pattern of children, as well as the effectiveness of the plan of treatment in maintaining a high degree of metabolic control.[21] By years of trial and error, we have observed that as long as the insulin requirement remains less than about 0.70 U/kg/day, it is not only possible but also practicable to maintain an essentially euglycemic state with freedom from glycosuria and hypoglycemic episodes.

However, when the exogenous insulin requirement begins to exceed about 0.70 U/kg/day, we have found it necessary to modify our objective of management to a slightly lower degree of control. At this time we reinstruct the parents and children to accept minimal transient glycosuria to widen the threshold of safety for preventing hypoglycemia. Recent studies of Liljenquist *et al.*[22] have confirmed our assumption that a slight overdosage of exogenous insulin, during the period of partial remission, would be compensated for by suppression of endogenous insulin. These authors demonstrated inhibition of endogenous secretion by exogenous insulin in normal men by use of the C-peptide assasy.

Our diabetic subjects were tested under standardized conditions and, as documented by HbA_{1c} determinations, they were in a higher degree of metabolic control than the patients reported by Gonen *et al.*[17] Although we anticipated an interrelationship between the exogenous insulin requirement and the endogenous insulin reserve, we were surprised to find such a consistent stepwise decrease in C-peptide levels as the exogenous insulin requirement increased *(Table II, Figure 3)*. The sensitivity of the assay (0.10 ng/ml) done in Rubenstein's laboratory with antiserum supplied by Binder and Faber with a low cross reactivity to proinsulin no doubt accounts for most of the consistency of the results observed.[9] In addition, the diabetic subjects received more purified pork insulin known to decrease the production of antibodies.[23] This study provides additional evidence that early and adequate insulin replacement is important to restore beta cell

(Continued on page 353)

Percutaneous Transluminal Angioplasty

Therapy for Vascular Occlusive Disease of the Lower Extremities

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Percutaneous transluminal angioplasty was used in 22 patients with vascular occlusive disease of the lower extremities. Short term results based on clinical symptoms and noninvasive studies are analyzed and compared. Continued evaluation with objectively documented results is needed to define the role of transluminal angioplasty and to determine long term results.

PERCUTANEOUS transluminal arterial dilatation was first described by Dotter and Judkins[1] in 1964 using coaxial teflon catheters to gradually dilate atherosclerotic lesions. This technique was not widely accepted because of local complications occurring at the puncture site when such large catheters (12 F) were inserted. Also, the high shear force generated on the arterial wall as these large catheters passed through the vessel raised many concerns. In 1974, Gruntzig[2] modified this technique by using a small diameter catheter with a balloon tip which could be inflated within a stenotic segment of artery compressing the atherosclerotic lesion and increasing the diameter of the vessel. Unlike the Dotter technique, the Gruntzig method could be used to dilate arteries remote from the arterial puncture site making dilatation of renal, iliac, popliteal, and coronary arteries possible. This technique has since achieved wide clinical application. Reported here is our experience with percutaneous transluminal angioplasty used in the treatment of arterial occlusive disease of the lower extremities.

Materials and Methods

Percutaneous transluminal angioplasty was performed on 22 patients for symptomatic arterial occlusive disease from May 1980 to December 1981 at St. Luke's Hospital of Kansas City. There were 14 men (64%) and eight women (36%). The age range was 44-89 years with a mean age of 62 years. All patients were white. Twenty patients (90%) were smokers and six (27%) were diabetic. Twelve patients (54%) had hypertension and only one (4%) had hyperlipidemia (Type II B). Nineteen patients (86%) presented with claudication and three (14%) presented with rest pain. Eight patients (36%) had previous vascular operations (four had coronary artery bypass, one each had bilateral carotid endarterectomy, above knee amputation, femoral-popliteal bypass, and brachial embolectomy).

All patients were initially evaluated with a careful history and physical examination. Segmental arterial systolic blood pressure measurements with ankle-arm index were done in all but one patient prior to angioplasty. Dynamic exercise treadmill testing with measurement of ankle-arm index was done in 18 patients prior to angioplasty. The four who were not tested were unable to walk on the treadmill because of previous amputation, severe congestive heart failure, metastatic cancer, and severe osteoarthritis. The standard treadmill exercise test involved walking on a ten degree incline at 2.5 miles per hour. The test was terminated when severe claudication developed, or at five minutes if the patient remained asymptomatic. Immediate post exercise ankle-arm index was recorded at the termination point. Arteriography was done in all patients.

After angioplasty, segmental blood pressures with ankle-arm index were repeated in 18 patients. Treadmill testing was repeated in 12 patients.

In February 1982, 18 patients were contacted by telephone and questioned about their current clinical symptoms and how they compared to their pre-angioplasty symptoms. Two patients had died but information was obtained from the family or from hospital records at the time of death. Two patients could not be contacted. Four patients had undergone arterial bypass on the involved extremity and were not requested to return for repeat non-invasive Doppler studies. Fourteen patients were asked to return

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for repeat noninvasive Doppler studies, and 12 complied.

Technique

An ultrasonic flow detector is placed over an ankle artery and a blood pressure cuff is placed around the calf for determination of ankle blood pressure before, during, and after the procedure. The ipsilateral groin is prepped and draped and the femoral artery is punctured with a 20 gauge angiographic needle. A large guide wire (0.038 inch diameter), which is easier to advance through a diseased vessel, is then passed into the artery through the needle and the needle removed. The guide wire is advanced through the stenosis and a six french angiographic catheter is passed over the guide wire. The large guide wire is replaced with a small guide wire (0.018 inch diameter) so that pressures above and below the stenosis can be measured through the catheter. Five thousand units of heparin are given intra-arterially. Spot films are taken to localize the stenosis and a hemostat is attached to the drapes to mark the stenosis. The angiographic catheter is removed and the balloon catheter is passed over the guide wire and positioned within the stenotic segment. The balloon is inflated and dilatation is carried out at a pressure appropriate for the size of the balloon and held for 30 seconds. A followup film is taken after injecting contrast material through the balloon catheter to check the result. Pressures above and below the stenosis are measured again. The dilatation is repeated until there is less than a 10 mmHg gradient across the stenotic area. The balloon catheter and guide wire are removed, and pressure is held on the puncture site for 20 minutes.

Patients are routinely given aspirin, 325 mg, twice daily and dipyridamole, 25 mg, three times daily for two days before the procedure. This is maintained indefinitely following angioplasty.

Results

Twenty-two patients underwent percutaneous transluminal angioplasty of the lower extremities during a 20-month period. The length of followup ranged from 1 to 19 months with a mean followup time of 9.4 months. The length of hospitalization for evaluation and treatment ranged from 2 to 43 days with a mean of 7.2 days. The pre-angioplasty hospital stay ranged from 1 to 17 days with a mean of 3.1 days. The post-angioplasty hospital stay ranged from 1 to 16 days with a mean of 3.7 days.

Fifteen patients underwent iliac angioplasty, six patients had femoral and one patient had popliteal angioplasty.



Figure 1. Graph showing pre-angioplasty, post-angioplasty, and late static ankle-arm indices with length of followup in 12 patients.

Fourteen patients (64%) had an improved dorsalis pedis pulse after angioplasty while eight (36%) remained unchanged.

Three patients (14%) underwent angioplasty for treatment of rest pain. All obtained relief immediately. One patient maintained this relief to followup at five months but did not return for ankle-arm index measurement. One patient had recurrent rest pain eight months later and underwent below knee amputation nine months after angioplasty. He subsequently died of pneumonia one month later. The other patient had continued relief of rest pain and showed an increase in claudication distance from two to ten blocks 19 months later. His static ankle-arm index had increased 0.43 immediately after angioplasty. At followup, the static ankle-arm index was 0.16 higher and his ankle-arm index after exercise was 0.19 higher than the pre-angioplasty levels.

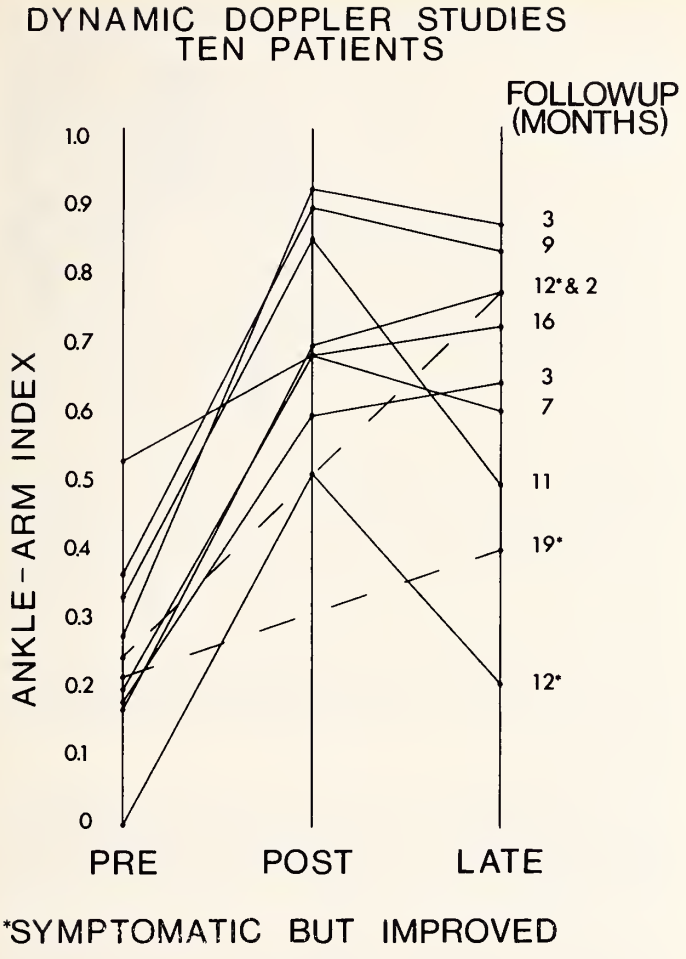


Figure 2. Graph showing pre-angioplasty, post-angioplasty, and late post-exercise ankle-arm indices with length of followup in ten patients.

LATE CLINICAL RESULTS - 22 PATIENTS

SUCCESSFUL		
ASYMPTOMATIC	12	
IMPROVED	3	
DISTAL BYPASS	2	
	17/20	85%
UNSUCCESSFUL		
BYPASS	2	
AMPUTATION	1	
	3/20	15%
LOST TO FOLLOW-UP	2	

Figure 3. Late clinical results of angioplasty for vascular occlusive disease of lower extremities in 22 patients.

Nineteen patients underwent angioplasty for treatment of claudication. Claudication distance ranged from ten feet to one-fourth mile. All patients improved initially following angioplasty. At followup, nine patients were asymptomatic. Four patients had undergone femoral-popliteal bypass — two for distal disease and two for unsuccessful angioplasty. Three patients remained symptomatic but had noticeable clinical improvement. One patient died 13 months after angioplasty from an intracerebral hemorrhage following carotid endarterectomy, and two patients were lost to followup.

Late followup Doppler studies were performed on 12 patients, all of whom were found to have a resting ankle-arm index higher than the pre-angioplasty level *(Figure 1)*. A comparison of the resting ankle-arm index in the immediate post-angioplasty period with the late followup period revealed little or no change in five patients, while five patients showed a decrease in ankle-arm index ranging from 0.26 to 0.30. One patient showed an increase in ankle-arm index of 0.14, and one patient did not undergo a post-angioplasty Doppler study.

Exercise testing using the standard treadmill stress was performed in 18 patients prior to angioplasty and demonstrated a marked decrease in the immediate post-exercise ankle-arm index in 17 patients *(Figure 2)*. The test was repeated immediately following angioplasty in 12 patients, all of whom were able to walk for a longer period of time on the treadmill and were found to have a higher ankle-arm index at the termination of the test than prior to angioplasty. The test was repeated in ten patients at late followup, all of whom maintained an ankle-arm index greater at the termination of the test than the pre-angioplasty level, but five patients demonstrated a decrease in ankle-arm index when compared to the immediate post-angioplasty level.

The degree of stenosis of the lesions that were dilated ranged from 75 to 95 per cent stenosis. Dilatation was not attempted in any vessel that was totally occluded. The length of stenosis that was dilated ranged from one to five cm. Twenty-one patients (96%) had technically satisfactory dilatations with the degree of remaining stenosis ranging from 0 to 30 per cent. One patient had a less than satisfactory dilatation (4%), with a 95 per cent lesion being dilated to an 8 per cent lesion. All patients, however, improved clinically after the procedure.

At followup, 20 patients were available for evaluation of late clinical success *(Figure 3)*. Fourteen patients (70%) were considered late clinical suc-

cesses because there was no limitation to their daily activity and no rest pain. Two of these had iliac angioplasties and had undergone femoral-popliteal bypass for distal disease. Three patients (15%) were considered improved because their walking distance had increased although they were still limited by claudication. Three patients (15%) were considered late failures. One of these subsequently had an amputation nine months after iliac angioplasty and two had femoral-popliteal bypass three months and six months respectively following superficial femoral angioplasty.

Morbidity and Mortality

There were seven (32%) complications among 22 patients. Four (18%) were minor complications which included two puncture site hematomas and two scrotal hematomas, all of which spontaneously resolved. There were three (14%) major complications. One patient developed a cold foot immediately following angioplasty and was placed on intravenous heparin for 24 hours. This rapidly improved and ultimately resolved two days later. One patient formed a clot proximal to the lesion as the guide wire was reinserted. The clot was dissolved with intra-arterial streptokinase, and the angioplasty was completed. The patient was maintained on intravenous heparin for 36 hours and then had repeat arteriography. Clots were seen at the puncture site. Heparin was restarted and the patient was maintained on coumadin for three months without permanent sequelae. The other patient developed pain in two toes after angioplasty, presumably from distal embolization. He was heparinized for 72 hours, and the pain resolved.

There were two deaths (9%) in 22 patients, neither related to the procedure. One died from aspiration pneumonia following a severe stroke ten months after angioplasty. The other died from an intracerebral hemorrhage one week following carotid endarterectomy and 13 months after angioplasty.

Discussion

Percutaneous transluminal angioplasty has been successful in alleviating symptoms of claudication and rest pain in selected patients. The technique has not been in use long enough to obtain conclusive long term results.

The iliac artery has been the most suitable vessel for this procedure. O'Mara *et al.*[3] reported an initial clinical success rate of 94 per cent, and a 100 per cent success rate at 7.1 months mean followup in a group having iliac angioplasty. Katzen *et al.*[4] had a 95 per cent initial success rate with 94 per cent successful at one year. Van Andel[5] reported a 92 per cent success rate at 33 months mean followup. Spence *et al.*[6] had initial success in 92 per cent, 93 per cent at one year, 82 per cent at two years, and 79 per cent at three years followup.

Femoral-popliteal angioplasty has not been quite as successful clinically. O'Mara *et al.*[3] had a 74 per cent initial success rate in a group who had femoral-popliteal angioplasty, but this dropped to 56 per cent at 8.4 months mean followup. Katzen *et al.*[4] reported initial success in 89 per cent, with 88 per cent still successful at one year followup. Spence *et al.*[6] had initial success in 84 per cent with 79 per cent successful at one year and 75 per cent at two years followup.

The initial clinical success rate of our combined group of iliac and femoral-popliteal angioplasties was 100 per cent. At 9.4 months mean followup, 85 per cent were either asymptomatic (70%) or clinically improved (15%), and 15 per cent were failures. This compares well with most reports in the literature.[3-6]

It is important to be selective in choosing patients for angioplasty. Results are best when lesions are isolated and localized without diffuse disease or vessel occlusion.[6]

The static ankle-arm index is a reliable method to objectively document improvement and to follow the clinical course of patients. Dynamic exercise treadmill testing also gives objective information about the patient's functional capacity and correlates well with the static Doppler studies. It did not, however, correlate closely with the clinical results in many of our patients because of their quiescent lifestyles.

The clinical application of percutaneous transluminal angioplasty should include the team approach of a vascular surgeon and a vascular radiologist. The vascular surgeon's training and experience provide him with clinical judgment in vascular disease and a knowledge of vascular pathology essential for proper patient selection as well as the technical skills to deal with potential complications. The vascular radiologist's training and experience provide him with technical skills in the use of intraluminal catheter procedures and judgment and experience in their clinical applications. Both disciplines are needed for the proper and responsible application of this therapeutic approach. Patients treated by this method should be followed closely by one or both members of this team. Our late followup noninvasive studies show a trend toward decrease in ankle-arm index although this is not yet reflected

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Theories of Dysmenorrhea

An Historical Review from 1850 to 1979

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Editor's Note: Papers published in the Student Thesis series have been written by fourth year medical students at the University of Kansas School of Medicine. Those selected for publication have been recommended by the UKSM faculty.

DYSMENORRHEA is a problem that has long frustrated the medical profession. It has been divided into two forms: primary dysmenorrhea whose etiology remains unknown, and secondary dysmenorrhea in which pain is associated with uterine or ovarian disease and disappears when the primary problem is resolved. It is one of the few medical problems in which an organ, upon examination, seems perfectly normal yet when functioning presents with symptoms associated with pathologic conditions.

Primary dysmenorrhea — also termed essential, functional, spasmodic, or intrinsic dysmenorrhea — may encompass a number of symptoms. In its simplest form it is pain associated with menstruation. However, the term will be expanded here to include a syndrome known as premenstrual tension since there was no distinction made between the two problems before 1931 when Frank first described the syndrome. The symptoms considered will therefore include not only uterine cramps, but pelvic discomfort, nausea, vomiting, weight gain, headache, back pain, abdominal distention, fatigue, and a host of psychologic symptoms such as depression, irritability, and generalized emotional tension. Some physicians have also included increased sexual desire, although this symptom is rare.

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Dysmenorrhea has been recognized as a medical problem since the time of Hippocrates,[1] but it became the center of an extensive medical and political controversy in the latter part of the 19th century. The medical profession became interested in it as part of their pervasive concern with women's health.

Little knowledge has been gained in the past century regarding the causes of primary dysmenorrhea. A psychosomatic factor has not been established. Although the scope of drug therapy for symptomatic treatment has increased, and contemporary research studies are scientifically conducted, many of the old prejudices persist.

Writers of popular manuals on women's health, for example, often brushed over widespread, fatal diseases such as consumption and breast cancer in their eagerness to describe the myriad dangers emanating from the womb. As one woman physician noted, following what she termed the Marion Sims epoch, "the profession went mad in the direction of gynecological tinkering, womb prodding and probing, and woman was rarely permitted to have an ache or a pain referable to any other part of her anatomy." According to another woman physician, there was "a wholesale onslaught upon those innocent organs . . . removing them for nervousness . . . because

the husband requested it, removing them for everything but disease.''[2]

Jules Michlet in 1868 characterized the 19th century as the ''age of the womb.'' Much of the medical literature of the time caricatured women as victims of a number of female complaints. Women did suffer from a great many diseases that doctors did not understand, but the exaggeration was due as much to the social and political changes of the time as it was to medical reality.

One of the major social changes of the period was the movement of women for equality, which included increased educational opportunities. From this sprang the social controversy that involved dysmenorrhea. Since the etiology of dysmenorrhea was not understood, there were many theories as to its cause, some with more social than scientific backing. It is the purpose of this paper to review this medical controversy that began in the middle to late 1800s and show how it has affected the thinking of physicians on dysmenorrhea up until the present time.

In 1852, Dr. Henry Bennet outlined primary dysmenorrhea as a condition that occurred as a result of the constitution of the woman, accidentally, or as a temporary state associated with poor general health. Women with a constitutional predisposition to menstrual pain were felt to suffer more if they were fatigued, under stress or anxiety, or even for no apparent reason.

Dr. Bennet also felt that dysmenorrhea might occur ''accidentally'' in a woman who had never before experienced any problem as a result of overexcitement or fatigue. From these beliefs he concluded that the discipline of public schools might be too much for women with a constitutional predisposition for dysmenorrhea of a severe type.

Dr. Bennet was describing a problem that plagued relatively few women for he was concerned with ''those cases in which a very considerable amount of pain is experienced,'' and not the mild discomfort ''most females experience.''[3]

However his concern that school might prove too much for a select group of women was soon to be expanded to include all women. One of the primary figures who influenced the public as well as his fellow colleagues was Dr. Edward Clarke. In 1872, Dr. Clarke addressed the New England Women's Club in Boston on the subject of the relationship of gender to education. It was this brief unstudied lecture that set the spark to the question of the relationship between menstrual problems and education. In order to clarify his position and strengthen it, Dr. Clarke set down his thoughts in a book entitled *Sex in Education or a Fair Chance for the Girls*. He felt that the rigors of formal education were too much for young women to handle, that they were in essence trying to do two things at once — develop their sexual organs and get their education. It was his theory that ''brain work'' causes a number of cells to die; that for every principle learned, nerve cells die and must be replaced. This in turn takes energy away from the developing sexual organs which then remain immature for the rest of the life of the woman and cause her to suffer from such problems as dysmenorrhea, amenorrhea, and even sterility.[4]

What did he advise? Up until puberty women could be educated in the same manner as men, but upon reaching adolescence, girls required special curricula to allow them to develop properly. This would entail working only three-fourths of the month with no work or very little during the other fourth. Dr. Clarke cited the European school system for girls as proof that he was right. He claimed that in Europe, girls stayed at home when menstruating, thereby conserving their energies for the development of their bodies. Thus European women were not so ''pale'' as American women and did not suffer from the problems so often seen in the United States.

Case histories were the greatest part of his scientific proof. He related the stories of various of his patients who had been educated and had done well in school, but emerged virtual invalids due to the strain on their bodies. They suffered weakness and pain. One young woman was so disabled that she had to be institutionalized.

However, Dr. Clarke was not the only physician with a theory as to the cause of menstrual problems. Some doctors felt that they were caused by early abortion, but if so, why did so many unmarried women suffer? Another theory proposed that menstruation itself was a disease and such things as dysmenorrhea were symptoms of the disease. It was felt that menstruation was a pathological process for the following reasons: (1) It is a result of the interference with nature; (2) It causes most women unpleasant symptoms which no other physiologic process does; (3) It requires the application of an ''artificial appendage'' and no hemorrhage is natural; and (4) Vicarious menstruation and pregnancy are superior in ridding the body of excess blood which is the cause of menstruation itself. The solution to the problem — pregnancy.[5]

Although physicians did not agree on the causes of dysmenorrhea of the essential type, they did agree generally on the treatment of it and the circumstances that might aggravate it. Essential dysmenor-

rhea was also termed neuralgic dysmenorrhea. J. H. Kellogg felt that chlorosis, gout, rheumatic conditions, excessive ingestion of meat, sexual excess, and secret vice brought about the problem. The best treatment was for the patient to rest quietly in bed for a few days and take hot baths.

Dr. Kellogg agreed with Dr. Clarke that young women might permanently damage themselves by excessive mental taxation during menstruation. However, he felt that a moderate amount of work would not be harmful. He also wondered about women workers; he thought they should be protected during their periods.[6]

Although the ideas about menstruation discussed above were all advocated by scientific men, they were not backed by research into the field through documented study. In the 1870s, male physicians were feeling threatened by the push of women to join the medical field and thus they tried to use any means possible to stop the movement. Vern Bullough and Martha Voght[7] commented on this phenomenon:

> When the belief structure of the physician is threatened, even in fields outside of medicine, he often uses his medical expertise to justify his prejudices and in the process strikes back with value laden responses which have nothing to do with scientific medicine. Unfortunately, since he is assumed to speak with authority, his response, perhaps as he intended, has influence far beyond that of ordinary men. One of the best examples of this is the controversy over the physical disabilities of women which took place in the last part of the nineteenth century as women began to demand more education, greater political equality, and to challenge many of the male stereotypes about woman's place. Since medical practitioners were almost all men, and many of them were hostile to any change in the status quo in male-female relationships, they inevitably entered the struggle with what today appear not only as ludicrous arguments, but which even in the period they were writing were not based upon any scientific findings, and in fact went contrary to those findings.

One such scientific study that was studiously ignored was Dr. Mary Putnam Jacobi's *Question of Rest for Women During Menstruation* published in 1877.[8] She ran a scientific study of women and their problems with menstruation, including dysmenorrhea. The study included both women who had continued their education during puberty and those who had not. The purpose of the study was to determine the number of women who suffered from dysmenorrhea, how many were forced to stop their activities due to pain, whether the woman's occupation had any effect on the problem, and what inferences could be made from the answers to the former questions regarding a woman's capacity for work.

She concluded that 47 per cent of women suffer from dysmenorrhea and that women who were occupied suffered less than women who weren't. She found that rest would not prevent the onset of menstrual pain and felt that only in extreme cases should women rest during menstruation.

But this study did little to deter most physicians from continuing their crusade. Physicians who followed Clarke carried his position even farther than he had. T. S. Clouston, Edinburgh, Scotland, wrote a series for *Popular Science Monthly* in which he said that it was medically accepted that "the female organism is far more delicate than that of men; . . . it is not fitted for the regular grind that the man can keep up." Overstimulation of the female brain, he said, caused stunted growth, nervousness, headaches and neuralgias, difficult childbirth, hysteria, inflammation of the brain, and insanity. He admitted that his argument was not based on statistical fact but only on the observations of physicians of their own patients. He also stated that he hoped research in the future would gather the facts, while he ignored the Jacobi study.

A better publicized study by the Massachusetts Labor Bureau on the health of American college women based on statistical evidence did little to change the attitude of physicians. The results showed that only 17 per cent were in poor health and that this was not markedly different from the national average. John Dewey who analyzed the report concluded that worry over personal matters was more harmful to health than overstudy.

Yet during the same year that the report was released (1886), Henry Maudsley wrote that the concurrence of puberty and higher education meant that mental abilities were developed at the expense of physical well-being, and he cited Clarke for proof.

The 20th century brought many changes in medicine, but much of Clarke's thinking was still evident for years to come. In 1905, Dr. F. W. Van Dyke objected to the practice of sending young women to school at a time when they should be in the open air playing, or at the very least studying within their limits. He stated that the curricula of most schools are tailored to the scholar and put too much strain on the average girl:

> Every physician knows that the cramming process, in the vast majority of cases, when applied to mediocrity, is the most frequent cause of hysteria, neurasthenia, dyspepsia, astigmatism, and dysmenorrhea.

Van Dyke also worried that education might lessen passion in women making them poor wives and mothers, and concluded that education of women

should be tailored for the average woman who would have no need to learn Latin and the sciences. Thus no girl would be forced beyond her capacity and suffer from unnecessary physical ailments.[9]

Dr. R. W. Parsons supported Van Dyke in his paper in 1907 in the *New York Medical Journal*. He noted that American women had begun to set themselves up as competitors with men for academic honors. While he conceded that this was indeed a noble calling, he went on to say, "admirable as these qualities of mind may be, the belief is becoming to be more and more strongly held by those who are in a position to speak authoritatively on the subject, that parents and educators are setting up the wrong ideals for our girls."[10] It was postulated that, as a result of their striving to compete, women suffered physically since, "The delicate organism and sensitive and highly developed nervous system of our girls was never intended by the creator to undergo the stress and strain of higher education, and the baneful results are becoming more and more apparent as the years go by."

Physicians were also still struggling with the problem of discerning the etiology of primary dysmenorrhea and often disagreed on the correct treatment. One predominant theory was that dysmenorrhea was caused by poorly developed uteri — a theory that would support the followers of Clarke since they claimed that education did not allow young girls to develop properly. Treatment included surgical incision of the cervical os and mechanical dilation of the os with rubber tubes.

In 1903, Herman and Andrews postulated that it was not imperfect development of the uterus that caused the pain, but a small and rigid canal that hindered physiologic dilation and provoked painful contraction of the uterine body. They recommended dilation or surgical incision as the means of treatment.[11]

Yet as early as 1911, doctors began to wonder if the treatment was not too vigorous. Brettaur felt that surgery and dilation might have harmed many women, and suggested another form of treatment based on his own beliefs about menstruation and dysmenorrhea:[12] "It is a biological fact, known for centuries, demonstrated daily in the animal kingdom, that there exists a definite connection between the sexual organs and the nasal mucosa."

When the nasal mucosa was treated, he contended, dysmenorrhea disappeared. Simply place cocaine on the "genital spots" in the nasal area and you stop the pain of menstruation. To effect a permanent cure, cauterize the same areas. He refused to accept the suggestion of his colleagues that either the cocaine itself or the power of suggestion was what caused the cure.

A somewhat more widely held view was that the best way to treat dysmenorrhea was to improve the patient's general health as a preventive measure. For relief of pain purgatives, local warmth to pelvic organs, and bed rest were advocated.

This recommendation was to remain a part of the treatment of dysmenorrhea for many years to come. However it did not hinder the search for better forms of treatment. In the search for new treatments, it was essential to try to discover how and why the problem developed. Since there was nothing anatomically wrong with the organ causing the pain, it seemed that physiology, psychology, or a combination of the two, must provide the answer.

In 1916, Dr. J. W. Kennedy described dysmenorrhea as both a symptom and a condition in which the local problem was much influenced by the type of patient. He suggested that the high strung or nervous type of woman suffered from a condition of the pelvic organs that would be merely a normal menstrual flow in one of a less nervous make-up. He recommended the analgesic antipyrin, and hysterectomy in severe cases.[13]

By 1925 the situation had not changed much. Dysmenorrhea was considered an "inherent abnormality" of the organs of menstruation with pain caused by an underdeveloped uterus, a retroflexed uterus, or a psychoneurosis. A stenotic os was discounted as a factor in most of the literature of the time and therefore the practice of splitting the os surgically was no longer an acceptable treatment. Dilation and curettage, while condemned by Blair-Bell, was accepted by most physicians as a worthwhile treatment.

Yet no treatment gave a truly satisfactory cure in all cases. Some women were cured by dilation, some with various drugs or minerals, and some by pregnancy, but there still remained a goodly number of women for whom only hysterectomy would cure. By 1930, Dr. Norman F. Miller pointed out that dysmenorrhea was still as much a mystery as it was 50 years earlier. He reported a decrease in the incapacitating form of dysmenorrhea, but the overall incidence was the same as that reported in the Jacobi study of 1877 — 47 per cent.[8]

Yet one important change had taken place since the time of Jacobi — study no longer was thought to harm women. In fact, a more active life seemed to help solve the problem. Dr. Miller found that improved posture and muscle tone in college women decreased their pain during a three year period. This occurred in women who had little chance for rest;

indeed they had long irregular hours, bore the strain of study and examination, and still improved. But many physicians still felt that there was a large psychological component.[14]

In a discussion of Dr. Miller's study, Dr. T. L. Montgomery stated:

> The presence of a neurotic state of mind in most patients suffering with essential dysmenorrhea has been observed so universally that one familiar with a family's history and traits can usually prognosticate the occurrence of dysmenorrhea in its younger generation of females.

It was also during 1930 that a new term was coined — premenstrual tension — which includes all symptoms occurring before menstruation, including pelvic pain. Many psychologic problems, such as depression and irritability, were included. Thus the issue became even more clouded.

Dr. Clinton Smith, in his book *Woman Thru the Doctor's Spectacles,* did not feel that painful menstruation must be endured. In his opinion, dysmenorrhea was a local condition caused by a systemic problem that takes a "severe toll each month of nervous stability." He thought that urinary tract infections might cause dysmenorrhea, but the systemic problem was endocrine in nature. He advised women to study female physiology so that they might "be more tolerant of the efforts of her physician, who while doing his very best, will often be unable to immediately effect a complete solution to her problem."[15]

The same attitude of menstrual pain being hard to treat mixed with the hope that women will try to understand and bear with the doctor was expressed by Dr. Emil Novak in his 1937 book *The Woman Asks the Doctor*. Yet once again, a hint of Clarke's ideas lingers on:

> A factor which must always be considered is that of the general health and constitution of the girl. The girl who works too hard at school, who does not go in for recreation of any kind, who is perhaps thin, sallow, anemic and nervous, is much more likely to suffer with menstrual pain than the husky, out-door type of girl, although there are exceptions to this, as to other rules.[16]

Another theory that became prominent toward the end of the 30s was that primary dysmenorrhea had no physical component at all but was of psychic or mental origin. In 1939, Dr. C. Frederic Fluhmann, a professor of obstetrics and gynecology at Stanford University School of Medicine, stated that the origin and persistence of painful menses are unquestionably linked with the patient's state of mind:

> It is generally conceded that fear and anxiety play a very important part. The favorable response of large groups of patients to procedures which in reality belong to the realm of suggestion therapy also is indicative of an underlying psychoneurosis.[17]

Dr. Karl Menninger in the same year postulated that disturbances of menstruation were a result of unconscious repudiation of feminity by women. However, he did not claim that this was the only cause; he suggested it might also be organic, chemogenic, psychogenic, or a combination of the three.[18]

In 1944, Helene Deutch theorized that menstrual pain is a complication of the normal course of development. The psychologic basis may be birth fantasies, or the young girl might be embarrassed by the physiologic process and therefore choose to regard menstruation as an illness. This is also connected with the girl's awareness of her mother's monthly indisposition.[19]

Another theory, related to that of Dr. Menninger, was that of Drs. L. Snaith and B. Ridley in 1948 that maladjustment to the husband was the commonest and most potent dynamic factor in the production of neurosis that causes the somatic symptom of menstrual pain. It was felt that the patient with dysmenorrhea was in revolt against maturity. They recommended that the gynecologist work with the psychiatrist in treating such cases.[20]

A review of the literature of the 50s reveals a change in physicians' approach to dysmenorrhea. Psychologic factors still were felt to be involved in the etiology, but they no longer were thought to be the sole cause, and indeed were considered possibly to be secondary to an endocrine imbalance. Therefore the most logical treatment would be drug therapy.

The endocrine imbalance was thought to be a relative increase in estrogens due either to increased estrogen production or an insufficient progesterone production, or a combination of the two. The estrogen would then cause salt and water retention that brings the bloated feeling and weight gain so often described as part of premenstrual tension. This heaviness felt in the area of the pelvic organs might be interpreted as pain in emotionally unstable women. It was suggested that estrogen might also contribute to emotional instability by producing hypoglycemia which could produce psychic manifestations as well as weakness and fatigue.

Some even felt that psychogenic factors had nothing to do with the problem. A 1953 study was designed to relate the psychologic manifestations of premenstrual tension with physiologic observations through the study of EEG, hormonal and psychiatric tests. It was concluded that no strong case could be

made for the psychogenesis of premenstrual tension, and that factors such as mothers with menstrual symptoms influencing their daughters, faulty preparation for menstruation, or abnormalities in the psychosexual development of the patient were not the sine qua non condition for the occurrence of premenstrual tension.[21]

In other words, physicians were still as puzzled by the etiology of the disorder as ever. Psychogenic explanation became only an influential factor in the explanation. Hypercontractility of the uterine musculature was another theory as well as the angiospastic concept in which ischemia caused the pain. There was also the thought that the cause might relate to a temporary dysfunctional synergy between hormonal regulators, the myometrium, and receptors in the uterine vascular tree.

The treatment of dysmenorrhea did not change very much either. There were four possible courses of action: accept the temporary disability and resort to bed rest and heat; give pain relieving drugs; prevent or interrupt ovulation; or resort to surgery. Three of these treatments were old remedies. Pain relieving drugs had been given before, only now there was a greater choice of drugs to give. Only the third was new, but it could not be used on patients who wished to become pregnant, and the side effects of preventing ovulation through the use of sex hormones were unknown.

Medical interest also took another turn in the 50s — what effect did dysmenorrhea have on the work force? Since so many women had begun working outside of the home during World War II, and continued to work after the war, could it be that there was a financial loss with women in the work force? Were women taking off several work days each month because of menstrual difficulties?

As with all other aspects of dysmenorrhea, there was disagreement as to the answers to these questions also. Billings' study in 1953 found that absenteeism and work inefficiency loss of time varied according to the type of activity, being slightly higher in women with sedentary positions. Overall, it was found that there were 36-43 lost work days/month/100 women, and 62 decreased efficiency days/month/100 women.

Hamman in 1945 claimed that dysmenorrheic women were more sensative than others and that absenteeism because of dysmenorrhea was more than three times that resulting from the common cold. However, several other studies disagreed: *i.e.* Ewing (1931), Baetjer (1946), Miller and Behrman (1953), and Smith and Norris (1957).

Smith and Norris in 1957 found that dysmenorrhea among female employees causes less than 3 per cent of all sickness absence. They felt it was important only as a nuisance since it caused many problems with staff reorganization; however, they did wonder how many women called in sick and gave other excuses when dysmenorrhea was actually the problem.[15]

Svennerud's 1959 study found that absence because of dysmenorrhea represented only 2.5 per cent of all absence of office workers and 3.7 per cent of factory workers. In comparing absentee rates of dysmenorrheic and nondysmenorrheic women, no appreciable difference was found.[22]

Each of the above studies was carried out with great scientific care, a fact that must be contrasted with the reports of many physicians nearly 100 years earlier when Clarke was trying to keep women out of schools. It would seem then, that the influence of that period had pretty well dissipated by 1960 and that there would be little in the way of derogatory remarks about the ability of women to function in work and schools as well as men in the present day when women are once again striving for increased freedom. However, this is not the case, as many feminists will tell you. There is still a political element to the problem of dysmenorrhea. It was in 1970 that Edgar Berman, previously best known as the friend and physician of Hubert Humphrey, remarked that women cold not fill leadership roles because of the influence of their periodicity — *i.e.* their menstrual cycles and menopause.

The reason that such statements may still be made — although women have proven themselves capable to serve in Congress, hold executive positions and carry out research worthy of a Nobel prize — is that no one knows any more about the etiology of dysmenorrhea and premenstrual tension now than they did in the 1870s. Theories are abundant. Among them are many first suggested in earlier times that have not yet been either proved or disproved.

Estrogen-progesterone imbalance, excess progesterone angiospasm causing ischemia, and low levels of oxytocin are just a few of the ideas. The suggestion that dysmenorrhea is primarily psychosomatic is also prevalent. Among the psychogenic theories are Kantero and Widholm's 1971 report that unrecognized mimicking by daughters of their mothers is of primary importance.[23] In 1972, Berry and McGuire reported that the physiologic manifestations of premenstrual tension might be due to psychologic stress and included the role of suggestion, imitation of mother, negative attitudes toward feminity, and benefits of secondary gains as part of

(Continued on page 355)



Current
COMMENT

H. B. LINDSLEY, M.D., *Editor*

Malnutrition

Recognition and Correction by Enteral Nutrition

M. SHAPIRO, M.D.*; J. B. RHODES, M.D.† *and*
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MALNUTRITION reminds most physicians of primary Kwashiorkor (protein deficiency) or marasmus (protein-calorie deficiency), as found in starving children in Africa or Asia. However, malnutrition in the United States has been defined as a 10 per cent loss in usual body weight due to an inability to eat, digest, or absorb enough food to meet body requirements. By this definition, malnutrition is a common problem in American hospitals. Malnutrition varies in severity and can be subclassified as mild, moderate, or severe. A prospective survey at the Medical Center in 1978 revealed that 23 per cent of 147 consecutive patients had mild to moderate malnutrition. Severe malnutrition was present in 8 per cent of the patients; almost one in ten was severely malnourished. Malnutrition in midwestern hospitals is a significant clinical problem which has been underrecognized.

Assessment of Malnutrition

The severely malnourished or cachectic patient with muscle wasting and loss of subcutaneous fat is obvious. Mild to moderate degrees of malnutrition may be less obvious, especially in obese patients. *Table I* summarizes some tests that are readily available and sensitive in detecting malnutrition.[1] The serum albumin, total lymphocytes, and iron binding capacity are reduced as the body draws on serum and visceral protein to meet the body's need for protein and energy. These parameters may be low normal in patients admitted to the hospital and can plummet with an acute illness or fasting as borderline reserves are rapidly depleted. These routine tests are not specific for malnutrition, and a number of other factors may account for alterations in the levels.

Additional methods are available for assessing nutritional status. These include dietary history, triceps skinfold thickness (fat), arm circumference minus fat (muscle protein), skin tests, and the 24 hour urea nitrogen excretion.[2]

More sensitive tests exist for assessing early protein deficiency, but they are not yet commonly available. Retinol binding protein (half-life 12 hrs) and prealbumin (half-life one to two days) rapidly decrease in protein malnutrition. Serum albumin (half-life 19 days) and iron binding proteins (half-life eight days) decrease more slowly. Rapid turnover proteins are especially useful in following therapy because their concentrations rise more rapidly than albumin and iron binding protein.[3] These tests are available through large commercial laboratories.

Body Response

The body's response to increased energy and protein needs with a decreased intake of food is well known. Glycogen is depeleted in a few hours and the breakdown of plasma and visceral and muscle protein to amino acids begins. Some of the amino acids

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TABLE I
ASSESSMENT OF NUTRITIONAL STATUS

	Degree of Malnutrition		
	Mild	*Moderate*	*Severe*
% of usual body weight	<95	<85	<75
Serum albumin (gm/dl)	<3.5	<3.0	<2.5
Total lymphocytes (Tot WBC × % lymphs)	<1500	<1200	<800
Total iron binding capacity (mg/dl)	<300	<225	<150

are used in protein synthesis, but most are deaminated to enter the energy cycles. Liberated amino groups are converted to urea and excreted in the urine. Nitrogen output then exceeds nitrogen intake — the classical negative nitrogen balance. Intravenous glucose reduces this nitrogen loss by about one-half in moderately ill patients. After several days, an increasing amount of energy is derived from fat and the rate of nitrogen loss drops again. The ability to convert fat to energy is reduced in patients with infection or trauma. Therefore, protein malnutrition is the most common form of malnutrition in patients whether they are normal or obese, young or old.

Protein-calorie malnutrition affects a number of organ systems including the gastrointestinal (GI) tract. Malnourished patients are often anorexic and their sensations of taste and smell may be blunted. Stomachs may empty slowly leading to early satiety, nausea, and vomiting which further reduce food intake. The pancreas, small intestine, and liver are protein depleted and digestion, absorption, and protein synthesis are impaired. Wounds and incisions are slow to heal. Antibody production and macrophage function are suboptimal. In addition, fever, leukocytosis, and tissue repair place additional demands on protein and energy stores.

Nutritional Replacement

Mild malnutrition can be corrected by simply increasing the intake of normal food. Between-meal snacks or commercial supplements can be used, and the physician should encourage additional food intake. Moderate degrees of malnutrition in many patients will usually require more intensive therapy. Enteral nutrition is suitable as the nutritional therapy for about 80 per cent of patients with malnutrition. The major requirement for enteral nutrition is an adequate GI tract. Patients with organic obstructions, an ileus, or severe GI disease may not be appropriate candidates. Enteral nutrition has several advantages to total parenteral nutrition. Enteral nutrition is simpler and has fewer side effects. In addition, less nursing time is required and the cost is significantly lower.

Patients with severe malnutrition or hypercatabolic syndromes can be difficult to replete with enteral nutrition alone. In these cases, the peripheral veins can be used to provide an extra 1000 to 2000 kcals/day.[4, 5] In other patients, where daily requirements are several-fold above normal, parenteral hyperalimentation via a central vein may be required.[6]

Practical enteral nutrition is now available due to three recent advances. These include small soft enteral tubes; pumps for reliable and continuous infusion; and nutritive solutions of low viscosity to pass through the small tubes.

The small feeding tube is inserted through the most patent nostril. The patient swallows and the tube is advanced. Many tubes contain a stylet which is useful in comatose patients and helps in passing the pylorus. If getting the tube past the pylorus is a problem, intravenous metoclopramide (Reglan) should help.[7] An x-ray or fluoroscopy can document duodenal placement which avoids the possibility of vomiting and aspiration.

A number of pumps are available to assure a constant flow rate of 25-200 ml/hr. The constant perfusion does not interfere with sleep and the patient can eat, drink, and swallow medications with the feeding tube in place. Around-the-clock perfusion provides maximal efficiency in the use of the digestive and absorptive tract.

The large number of suitable solutions come in three basic types. The first type contains carbohydrates, fat, and protein in large molecular forms, resembling blenderized food. The proportions (kcals) are about 50 per cent carbohydrates, 30 per cent fat and 20 per cent protein, which is similar to a normal diet. These preparations exert few osmols and require pancreatic enzymes and small bowel digestion. Some of these solutions are too viscous for the small feeding tubes while others can be taken orally or via the tube. The second and third types of nutritive solutions are less viscous and can be pumped through the small feeding tubes. These solutions contain partially hydrolyzed starch which increases the osmols. The third general type contains smaller molecules such as free amino acids and is quite hypertonic. In general, the potential for overloading the GI tract increases with increasing osmols.[8]

TABLE II
ENTERAL NUTRITION: STRENGTH OF SOLUTIONS AND FLOW RATES

	Day 1	2	3	4
Solution strength	⅓	½	Full	Full
Flow rate (ml/h)	50	75	100	150
24 h intake				
Water (ml)	1200	1800	2400	3600
Energy (kcal)	400	900	2400	3600
Protein (gm)	15	35	96	140

TABLE III
ENERGY AND PROTEIN REQUIREMENTS OF PATIENTS

	Energy (kcal/kgm)	Protein (gm/kgm)
Maintenance		
Inactive	25	0.7
Active	35	1.0
Therapeutic — For Malnutrition		
Mild	40	1.5
Moderate	50	2.0
Severe	70	3.0

In mild malnutrition, the pancreas and small bowel are only minimally malnourished and can digest and absorb a normal food load. In moderate malnutrition, the malnourished GI tract takes several days to replete itself. *Table II* summarizes one method of preparing the GI tract to attain maximal digestive and absorptive efficiency. Liquid formulations are diluted with two volumes of water and perfused at a rate of 25-50 ml/hr. Powdered formulations are dissolved in three volumes of the usual amount of water and pumped at the same rate. If the perfusion goes well for 8-12 hours, the rate is increased to 50-75 ml/hr. It is well to increase the flow rate for 8-12 hours before proceeding to the next highest concentration. If diarrhea occurs, the GI tract has been overloaded, and the flow rate and concentration should be reduced to the last successful concentration and perfusion rate, and advanced again. In severe malnutrition, GI function is markedly impaired, and more dilute solutions at lower flow rates may be required initially.

Lactase and sucrase deficiencies are common in severe malnutrition and solutions containing lactose or sucrose are best avoided. Most nutritional formulations are excellent culture media for bacteria which can also cause diarrhea. Freshly opened or mixed solutions should be used within 8-12 hours.

When caloric intake approaches 2400-3000 kcals/day, the formulation should include 30-40 per cent fat. Large glucose or oligosaccharide loads (500 gm/day or 2000 kcals) have some disadvantages and problems. Large carbohydrate loads have a high osmotic effect and may cause diarrhea. They may elevate the blood sugar with glucosuria. High glucose loads are lipogenic, and a fatty liver may cause mild elevations in liver function tests. Large glucose loads also require more oxygen for metabolism and induce a hypermetabolic state, increasing temperature and heart rate. Finally, carbon dioxide production is increased and patients with chronic obstructive pulmonary disease may have problems exhaling the additional carbon dioxide.

Inclusion of fat in the diet keeps the energy supply up and helps avoid most of the problems associated with high carbohydrate loads. Fat is calorically dense and supplies more energy gram for gram (9 kcals/gm to glucose 4 kcals/gm). Fat has a low osmotic force when incorporated in normal bile salt micelles and prevents essential fatty acid (linoleic) deficiency.

When protein intake approaches 100 gm/day, increased urea production may elevate the BUN and some extra water may be required to reduce the prerenal azotemia.

Therapy

The amount of energy and protein required to correct malnutrition varies with a number of factors — age, sex, height and weight, as well as type, duration, and severity of primary disease. *Table III* is a general guide for maintenance and therapeutic nutritional replacement. Protein recommendations assume a good protein source such as milk, egg, meat protein, or amino acids. The total energy and protein requirements can be reduced about 20 per cent for patients 65 years and older.

Other nutritional products can be added to the usual preparations to tailor the nutrient replacement to the individual patient. Partially hydrolyzed starch can be added to increase carbohydrates which increases the osmols. Hydrolyzed starch plus fat in a 50-50 caloric mix provides extra fat calories with a lower osmolar load. Milk whey is a common protein additive; it contains 10 per cent carbohydrate as lactose, which may be a problem in lactase deficient patients. These additives can increase the caloric

TABLE IV
COMPLICATIONS OF ENTERAL NUTRITION

Complication	*Incidence %*	*Probable Cause*	*Correction*
GI Tract			
Vomiting	10-15	Slow gastric emptying	Tube in duodenum
Aspiration	1		
Diarrhea	10-20	Overload	Slow perfusion
		Infected diet	Culture diet
		Antibiotic	Discontinue
Metabolic			
Hyperglycemia	10-15	Glucose load	More fat
		Prediabetic	Insulin
Heart failure	1-5	Weak heart	Digitalis
		Fluid load	Conc. diet
Edema	20-25	Malnutrition	Nothing
		Fluid load	Diuretics

density from the usual 1 kcal/ml toward 2 kcal/ml and are useful in patients in whom a high fluid load is undesirable.

Water soluble and fat soluble vitamins are indicated in moderately malnourished patients, and are required when the total daily intake of the nutritional solutions is greater than 1500-2000 kcal/day. Folate, vitamin B_{12}, and vitamin K should be added. Extra iron is required by many patients. Trace minerals are usually present in the nutritional solutions in adequate amounts, but extra trace minerals can be added once every week or two in severely malnourished patients.

Complications

The complications of enteral solutions are easily detected and easily managed. *Table IV* summarizes the major complications. The first and potentially lethal complication is the infusion of hypertonic solutions into a stomach that empties slowly. The symptoms are epigastric fullness, followed by nausea and vomiting. If the patient is comatose or weak, aspiration of vomitus can be fatal. This is the primary reason why bolus injections of nutrient solutions are less commonly used today. This problem is avoided by infusing the formulations into the duodenum and starting with dilute solutions at low flow rates. If this problem is anticipated, the infusion can start with isotonic saline and glucose.

Diarrhea is less serious but annoying to patients and nurses. Early diarrhea is usually due to overloading a malnourished GI tract and can be corrected in a few days. Diarrhea out of proportion to the flow rate and concentration suggests an underlying defect in the digestive-absorptive mechanisms.

Hyperglycemia and electrolyte abnormalities may occur during the early replacement period. Increasing the fat content in place of the carbohydrate is helpful for hyperglycemia. Diabetic or prediabetic patients may require insulin. Heart failure is uncommon, but should be looked for, especially in elderly and severely malnourished patients. Edema is common and subsides with time as the nutritional status improves. Diuretics are not often needed.[9]

A number of other complications have been observed but their occurrence is generally less than 1 per cent. These unusual complications include clogging of the feeding tube, hypernatremia, hypercalcemia, essential fatty acid (linoleic) deficiency, esophageal erosions, and hyperosmolar coma.[9] Another annoying problem may be constipation because the tube feedings are low in residue, and fecal volume usually decreases. If the patient does not have a stool for three or four days, a tap water enema may be given to relieve constipation distress. Many patients pass a mushy stool every three to seven days and do not feel constipated.

Monitoring the Patient

The patient on enteral nutrition should be monitored closely for several days *(Table V)* and less closely as the patient stabilizes. The urine glucose should be checked two to three times a day to detect early hyperglycemia. Heart, lung, renal, and GI function should be assessed for several days. Daily volume and caloric intake should be recorded and the volume output measured as accurately as possible and correlated with daily weights. The increase in real body weight should be about 0.5-0.7 kg

TABLE V
MONITORING THE PATIENT ON ENTERAL NUTRITION

Initial — Daily for several days

Assess organ function
Heart-Lungs-Renal-GI

Vol and kcal intake

Body weight

Urine-glucose-tid
Serum-Elect-Glu-BUN

(1.0-1.5 lb)/day. More rapid weight gain is due to the accumulation of salt and water. Serum electrolytes, glucose, and BUN should be monitored daily until the patient stabilizes and then checked twice a week. Other serum profiles can be checked once a week. Correcting moderate degrees of malnutrition may take several weeks, and home enteral nutrition in the stable patient is easy.

Even though visceral protein is replaced, the patient may remain weak. Muscles must be exercised to rebuild the muscle protein. If the patient is unable to be up and about, active exercise in bed is required. When the patient's intake of normal food increases, the flow rate can be reduced or the feeding tube can be used overnight for extra calories and protein. When the patient is able to meet energy and protein requirements orally, the tube can be retired.

Unfortunately, nutritional replacement cannot help every patient. If death is near, and the primary disease is winning, nutritional replacement may offer no real benefit to the patient. In these instances, the feeding tube may be used for fluid, electrolyte, glucose, and drug administration. The enteral feeding tube is more convenient than trying to maintain intravenous lines for long periods of time.

While correcting malnutrition will not cure the primary disease, it will certainly enhance the patient's ability to respond to the primary disease. It will also reduce the incidence of confounding complications such as infections and increase the patient's strength. Improved nutrition should reduce morbidity as well as mortality.

Self-Assessment Questions

A 65-year-old woman lost her husband six months earlier. She had a poor appetite and lost 6.7 kg (61.2 to 54.5 kg) in five months. She then caught the flu with nausea, vomiting, malaise and cough, and after 11 days had lost another 4.5 kg. Her cough became productive of thick yellowish sputum with chest pain and a temperature of 39.4C. After three days, her appetitie is poor, she tolerates liquids, and has lost a total of 11.2 kg.

Directions: Pick the single best answer for each question.

1. This patient has lost 18-19 per cent of her usual body weight. Serum albumin is 2.9 gm/dl, and total iron binding capacity is 220 mg/dl. This patient's degree of malnutrition is *(Table I):*
 a. None
 b. Mild
 c. Moderate
 d. Severe
 e. Near terminal
2. The patient's weight is currently 50 kg. Her daily energy and protein requirements are *(Table III):*

	Energy (kcals)	*Protein (gms)*
a.	1250	35
b.	1750	50
c.	2000	75
d.	2500	100
e.	3500	150

3. Sputum cultured Staph aureus is found to be sensitive to ampicillin, which is started. The dietician reports that the patient is taking 2 liters of fluids, but is getting only 750 kcals/day. Her heart, lungs, kidneys, and GI tract are otherwise satisfactory. A small feeding tube is confirmed by x-ray to be in the duodenum. The hospital stocks a tube solution that contains 1 kcal/ml. The patient tolerates half-strength solution at 50 ml/hr. To correct her malnutrition, the flow rate and strength to deliver the required energy and proteins should be *(Table II):*
 a. 50 ml/hr; ⅓ strength
 b. 75 ml/hr; ½ strength
 c. 100 ml/hr; full strength
 d. 125 ml/hr; full strength
 e. 150 ml/hr; full strength
4. Common complications (10% or greater) of enteral alimentation are *(Table IV):*
 a. Vomiting
 b. Diarrhea
 c. Hyperglycemia
 d. Edema
 e. All of the above
5. Appropriate total care of the patient includes:
 a. Dealing with depression
 b. Treating the Staph pneumonia
 c. Nutrition support
 d. All of the above

(Answers on page 356)



Drug INFORMATION

JOYCE GENERALI, R.Ph., *Editor*

Drug Interactions Between Anticonvulsants

Interactions of anticonvulsants with drugs in other therapeutic classes have been extensively reported in the literature.[1, 2] Many review articles have been written on this subject, especially regarding phenytoin.[3, 4] Because many patients require more than one anticonvulsant for seizure control, the focus here will be on the interactions *between* anticonvulsants.

Phenytoin

Phenytoin, an enzyme inducer, decreases serum levels of carbamazepine, clonazepam, and valproic acid.[4-7] Conversely, phenytoin has been shown to increase phenobarbital serum levels.[4] Other studies indicate that phenytoin preferentially enhances the conversion of primidone to phenobarbital rather than to phenylethylmalonamide.[8] Thus, for a patient receiving primidone, phenytoin increases phenobarbital serum levels via two mechanisms.

Phenobarbital

Phenobarbital is another enzyme inducer that decreases serum levels of carbamazepine, clonazepam, and valproic acid.[5, 9] The effect of phenobarbital is variable. At lower therapeutic doses, phenobarbital can inhibit phenytoin metabolism.[10]

Carbamazepine

Carbamazepine not only induces the metabolism of other anticonvulsants via enzyme induction, but also induces its own metabolism.[11] Reports indicate that carbamazepine will reduce serum levels of clonazepam, ethosuximide, phenytoin, and valproic acid.[7, 12-14]

Valproic Acid

Valproic acid inhibits the metabolism of phenobarbital in most patients.[15] In one report, 11 of 13 patients required decreased dosage of phenobarbital (average dose reduction required was 46%).[16]

The effects of valproic acid on phenytoin are complex. Initially, valproic acid displaces phenytoin from protein binding sites, resulting in increased free phenytoin serum levels and increased metabolism of phenytoin due to the increase in free phenytoin. However, the total serum levels are decreased. After several weeks, valproic acid may inhibit phenytoin metabolism allowing phenytoin serum levels to return to that prior to valproic acid therapy.[17] Usually *no* dosage adjustments are required, unless the patient is at the upper limits of the therapeutic range for phenytoin. Loading doses of phenytoin should be administered with caution to patients receiving valproic acid due to the possibility of transient phenytoin toxicity.[18]

Valproic acid has also been reported to increase serum levels of ethosuximide and possibly clonazepam.[19]

Clonazepam and Ethosuximide

Very limited information is available concerning the effects of these drugs on other anticonvulsants. One report postulates that each drug independently decreases carbamazepine levels.[5] Conflicting evidence exists on the effect of ethosuximide on phenytoin. One author has reported that ethosuximide may increase phenytoin serum levels, while another

(Continued on page 353)

Socio-ECONOMICS

Intermediate Care for Mental Health Under Medicaid

A RECIPIENT in need of an intermediate level of care is one who is under observation, treatment and care for illness, disease, injury, or a handicapping condition which requires medical supervision and nursing care carried out under a physician's direction, but does not require 24-hour professional supervision.

ICF-MH (previously ICF-MI) Care

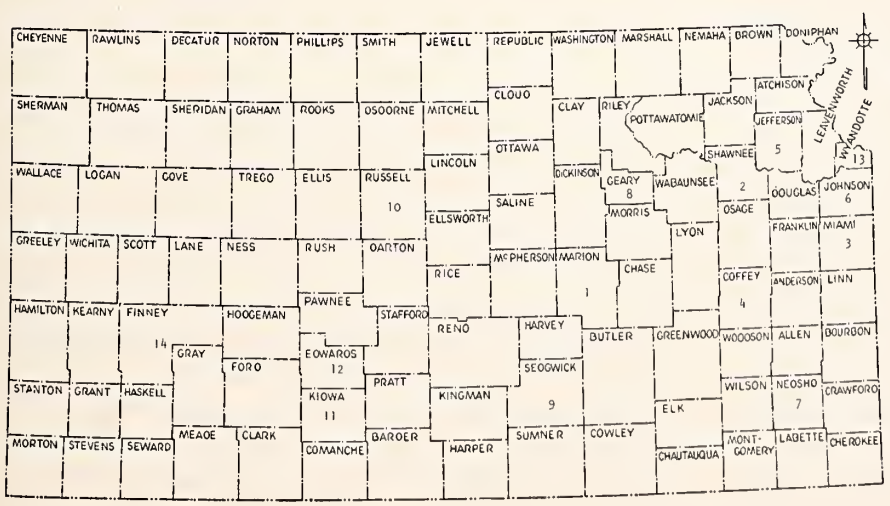
A recipient in need of an Intermediate Care Facility for Mental Health (formerly Mental Illness) level of care is one who requires 8 hours/day, 7 days/week licensed nursing supervision for on-going observation, treatment or care for long term mental illness. This individual requires maintenance/rehabilitation, nursing, social, behavior control activity, and dietary services which are routine in nature in the sense that they can be performed by supportive personnel and supervised by the appropriate licensed/certified professional or technical mental health staff. These facilities are staffed to treat physical as well as emotional problems. A community mental health center or private psychiatrist should be available if a resident should experience behavior problems.

The activity program should meet the needs of the mentally ill through therapeutic and recreational normalization processes; for example, learning to go into the community and socialize appropriately, relearning old skills, learning new skills (self-care, hygiene, grooming), learning to structure own time, etc.

Community Mental Health Centers or private psychiatrists are also available to direct planning and implementation of treatment plans for residents, make evaluations/assessments as needed, provide individual psychotherapy when indicated, provide medication monitoring, and provide training to staff of the facility.

Determination for Transfer from ICF to ICF-MH

To be transferred from ICF to ICF-MH, residents in an ICF must meet State approved Title XIX criteria for having a primary medical need. If they do not meet such criteria but based on documentation, interview, and diagnosis they do meet the criteria for ICF-MH placement, such a move will be recommended and payment will be terminated at the initial ICF. The recipient may, however, choose to pay for his own care and remain in the ICF.



APPROVED INTERMEDIATE CARE FACILITIES FOR MENTAL HEALTH

Facility Name/Location

1. Autumn Manor, Florence (Marion)
 Westview Manor, Peabody (Marion)
2. Countryside Health Center, Topeka (Shawnee)
 Howe Care Center, Topeka (Shawnee)
 The Oaks, Topeka (Shawnee)
3. Osawatomie Rest Home, Osawatomie (Miami)
 Medicalodge of Paola, Paola (Miami)
4. Sunset Manor, Waverly (Coffey)
5. Valley View Home, Valley Falls (Jefferson)
6. De Soto Rest Home, De Soto (Johnson)
 Edwardsville Manor, Edwardsville (Johnson)
7. Autumn Manor, Chanute (Neosho)
8. Valley Vista Care Center, Junction City (Geary)
9. Heartland Rehab Center, Wichita (Sedgwick)
10. Ala Fern, Russell (Russell)
11. Siesta Home, Haviland (Kiowa)
12. Medicalodge of Kinsley, Kinsley (Edwards)
13. Bryant-Butler-Kitchen Nursing Home, Kansas City (Wyandotte)
14. Briar Hill Manor, Garden City (Finney)

The President's Message



Dear Colleague:

The Annual Meeting in Lawrence was a success and the Douglas County Medical Society and Auxiliary are to be commended on their outstanding work.

Your State medical society is embarking on innovative and exciting programs. The House of Delegates endorsed a concept that will make this Society, with your help, a very active and meaningful organization. Committee structure has been changed to better address pressing issues. New committees will be formed for specific purposes. Involving all members is a goal that we accept as a challenge.

The legislative interim study committees will be involved in many medical issues, including professional liability and physician extenders. Many of us will be involved in these hearings. Medical Society committees are beginning to meet to develop appropriate testimony and response to these issues with state agencies that are involved.

Be alert for announcements and information in the form of pamphlets, booklets and seminars. We are a most effective organization when all of us are involved in the process. But we, indeed, need your help.

President



The Evolutionary Revolutionary

There would be little argument with the proposition that medicine has been — and still is — deep in revolution. In these days of hyperbole, however, the literal truth of the assertion must be emphasized since that perfectly good and reasonably explicit word has been bandied about by the hucksters and media generally so that the benumbed public sensorium reflexly distorts its meaning, applying it where it is not fact and diminishing it when it is.

Military revolutions are generally recorded in terms of decisive battles or outstanding personalities or even some idealistic purpose behind it all, and the medical revolution has all of these elements. A case can be made that the medical condition is only a part and reflective of the larger world situation. The latter, however, seems not so much a state of revolution as one of constant and moveable crisis. In our personal understanding, a revolution embodies a series of contentions between the established (however briefly) system and forces attempting to displace it in favor of a different and better one (at least in the opinion of those forces). However much this might seem to fit, the tumultuous experience of the 20th Century seems rather to be one of profound education-communication-transportation changes in which the goal is not so much revolution as acquisition.

Obviously, intrinsic medical progress has contributed to the upheaval in various ways — increasing longevity with consequent increases in population (particularly the very old, the very young, and the reproductive female segment, all of which were highly vulnerable in earlier times), the concept of replacement parts both natural and artificial with implications of immortality, fertility control both up and down, the limitation and sometimes control of infectious diseases, the transformation of Rube Goldberg fantasies to technical reality, the sudden discovery of health and the frenetic attempts to make up for all the sloth of ages past — all of the demographic, economic, religious, and psychologic complexities begotten by them. Within the medical province, these elements have indeed both produced and derived from a revolutionary atmosphere as previously held positions are forced to accommodate to the unbelievable — even unassimilable — progress.

We have no intention of trying to catalog all of the entities that have roles in the revolution — participants, or even observers who are too close, are not in a position to know a revolution's full character. Rather, it is to consider (again) one of the components that has moved into prominence in recent years, bringing with it prospect and challenge — genetics. It would be proper to qualify that as *medical* genetics and further, *human* medical genetics since plant and animal husbandry have been at it for a long time and, in fact, the heartening progress in human studies has been enhanced by their groundwork. It is obviously incorrect to speak of genetics as new — it is as old as life and, medically speaking, the genetic components are so pervasive as to be difficult to confine to a concise form. Even as taxonomic efforts progress, its manifestations turn into various channels — pediatrics, obstetrics, dentistry, orthopedics — literally every branch of medicine relates ultimately to genetics, not to mention the sociologic impacts of population genetics and the industrial potentialities of molecular biology. Compared with the wealth of pedigree information accumulated from earliest times, "pure" medical genetics is still tooling up and — to switch metaphoric horses — riding off in all directions.

Popular treatments generally date the "birth" of modern genetics from the efforts of Mendel and Darwin and Galton — perhaps that should be referred to as "conception" since a sort of gestation period was brought to fruition by the climatic delivery of the Watson-Crick double helix (which has done for genetics what Kekule did for organic chemistry in providing a working model). But even this

would not have contributed so much had not efforts of others been exposing the chromosomes and stripping them down to their underwear in search of the characteristics to match up with the human results — not to mention the biochemists who have been measuring indirectly the impact of genes too small to demonstrate visually. Practical applications of genetic features (blood grouping and tissue typing, for example) have meantime focused on the peculiarities of the infinite variety of human forms, emphasizing that however handy it is to be able to observe the chromosomes in their Dance of Life, they are crude aggregates of innumerable genes whose behavior must still be shown in round-about ways.

So we try to analyze their expression in human forms as they take their places in the respective medical departments and invade the privacy of the genes by laboratory police action to detect the criminal elements and rehabilitate them. But the resulting diagnostic and therapeutic and technologic upheavals will be accompanied by a greater one, one which seems destined to be self-perpetuating — the impact on the moral, ethical, and philosophic structure of our times. Since such convulsions will not be contained to be released in easily manageable forms for our convenience, we have no choice but at least to anticipate them and work toward adaptation of our pedestrian psyches to jet-age, computer-age, DNAge problems.

Since the phenotypic features of a given case have dictated its management, its genetic basis, even if known, has usually been assigned to the academic area — or dismissed with the short-sighted truth that it couldn't be changed anyway. But the prospect of genetic engineering (and here we move cautiously between the factual potentialities and giddy excesses of science fiction) now deny this comfortable dismissal. There will be a myriad of possible directions but each in its own way will confront us with the basic question: Is this a right and proper direction to go? Who says? At what cost? Who pays? What of the inevitable errors and misapplications? Will malpractice begin in the test tube (which it has already)? (How much easier to pose involved questions to promote an aura of wisdom than try to answer them!)

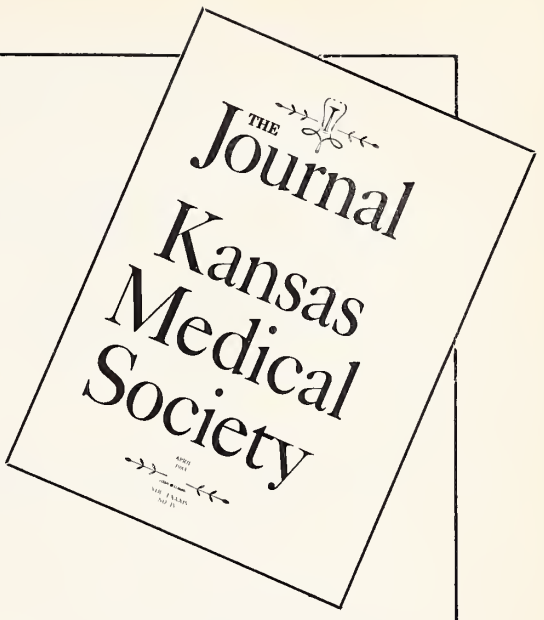
A public nourished on the dramatic and sensational aspects of genetic potential for changing lives may understandably have trouble distinguishing between the "mad scientist" aspect and the legitimate development of altered genes designed — but not guaranteed — to have a salutary effect on individuals specifically or the race generally. A world fed on a steady diet of marauding nuclei or the noxious byproducts of their indispensable comforts can perhaps be excused for suspecting that in the genetic effort, there is more than just the possibility of unknown and devastating results. Because of this, it will accept some things that are false and futile while they fear and reject others that are worthy. Even in our own medical world, comments such as this must guard against quoting or assessing the medical body as a unit for it already includes diverse attitudes as broad and varied as those of the public (with the slimmest margin of expertise to justify a louder voice).

Early indications of these problems have already emerged: the accusation of intended genocide in attempts to identify those at risk in racially concentrated disorders (sickle cell anemia, for example), disclosure of sensitive personal information in familial problems (as in Huntington's disease), revelation by genetic studies that a presumed parent cannot be the biologic parent, the many questions raised by prenatal diagnosis (even whether and when justified), to touch on only a few. And all are rendered more problematic by the newness of the efforts, the recruitment of the several disciplines that ultimately enter into the decisions, the continuing (although, it is hoped, decreasing) risk of error, and by no means the least, the problem of transmitting the information regarding highly charged and complex matters to individuals without technical background but with strong emotional interferences in reception.

The positive side, however, is that the same prospects and challenges have brought out strong social efforts to meet the complexities and provide a rational service geared to the current knowledge. Interest stimulated by the nature of the problems and by examples of successful efforts that have gone before — in other medical areas such as drug abuse more than genetics — has brought subjects and families together in the so-called "self-help" milieu. Not only have these been a healthy and productive focus of mutual support for those involved but a strong source of educational efforts to the public. Moreover, they are in a good position to maintain liaison with developments in the various fields, providing enlightened information on their status and progress, diminishing sensational overexpectations and misrepresentations.

We hope these are signs that mankind is mature enough to bring on the genes because they are here — ready to solve problems and to create them. — *D.E.G.*

Information for authors . . .



Manuscript Preparation

Manuscripts must be typewritten, double spaced (all text and references), leaving wide margins. Submit the original plus one copy if possible.

Titles will be reconstituted into a three-word headline with a descriptive blurb line.

Summary: All manuscripts should include a short, factual abstract.

Author Responsibility: The author is responsible for all statements in the manuscript, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

If more than one author is listed, one should be designated to whom correspondence is to be addressed. A copyright protection form will be sent to the corresponding author for signature.

Galley Proof: To make extensive changes in an article after it has been set in type requires an additional cost which could exceed the original. The galley proof is for correction of *ERRORS;* rewriting of material should be done prior to submission to THE JOURNAL. Authors are strongly urged to carefully check manuscripts prior to submission, and to check galley proof for typographical errors that could result in inaccurate information or changed meanings.

Drugs should be referred to by their generic names; trade names may be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references *should be typed double spaced,* and should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation — *not* alphabetically. All references must be numbered consecutively, and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material that cannot be set in type — such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) — must be identified on the back as to figure number, author's name, and an arrow indicating top. They must be referred to by number in the text, and should be numbered in order of referral using Arabic numerals. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words. Such materials should be submitted in camera-ready form, *i.e.*:

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high-contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost. Photos showing persons' faces will be published only with the eyes masked or with written permission from the subject.

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CONTRAINDICATIONS: ASPIRIN: Allergic or idiosyncratic reactions to aspirin or related compounds. MEPROBAMATE: Acute intermittent porphyria; allergic or idiosyncratic reactions to meprobamate or related compounds, e.g. carisaprodol, mebutamate, or carbromal.

WARNINGS: ASPIRIN: Use salicylates with extreme caution in patients with peptic ulcer, asthma, coagulation abnormalities, hypoprothrombinemia, vitamin K deficiency, or those on anticoagulants. In rare instances, aspirin in persons allergic to salicylates may result in life-threatening allergic episodes.

MEPROBAMATE: DRUG DEPENDENCE: Physical and psychological dependence, and abuse have occurred. Chronic intoxication from prolonged ingestion of, usually, greater than recommended doses is manifested by ataxia, slurred speech, and vertigo. Therefore, carefully supervise dose and amounts prescribed and avoid prolonged use, especially in alcoholics and others with known propensity for taking excessive quantities of drugs. Sudden withdrawal after prolonged and excessive use may precipitate recurrence of preexisting symptoms, e.g. anxiety, anorexia, or insomnia, or withdrawal reactions, e.g., vomiting, ataxia, tremors, muscle twitching, confusional states, hallucinosis, and, rarely, convulsive seizures. Such seizures are more likely in persons with CNS damage or preexistent or latent convulsive disorders. Onset of withdrawal symptoms occurs usually within 12 to 48 hours after discontinuation; symptoms usually cease within next 12-to-48-hour period. When excessive dosage has continued for weeks or months, reduce dosage gradually over 1 to 2 weeks rather than stop abruptly. Alternatively, a short-acting barbiturate may be substituted, then gradually withdrawn.

POTENTIALLY HAZARDOUS TASKS: Warn patients meprobamate may impair mental or physical abilities required for potentially hazardous tasks, e.g., driving or operating machinery.

ADDITIVE EFFECTS: Since CNS-suppressant effects of meprobamate and alcohol or meprobamate and other psychotropic drugs may be additive, exercise caution with patients taking more than one of these agents simultaneously.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during first trimester of pregnancy, has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at time of institution of therapy should be considered. Advise patients if they become pregnant during therapy or intend to become pregnant to communicate with their physicians about desirability of discontinuing the drug.

Meprobamate passes the placental barrier. It is present both in umbilical cord blood at or near maternal plasma levels and in breast milk at lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breastfeeding patients, consider the drug's higher concentrations in breast milk as compared to maternal plasma levels.

USAGE IN CHILDREN: Keep preparations with aspirin out of reach of children. Equagesic®-M is not recommended for patients 12 years of age and under.

PRECAUTIONS: ASPIRIN: Salicylates antagonize uricosuric activity of probenecid and sulfinpyrazone. Salicylates are reported to enhance hypoglycemic effect of sulfonylurea antidiabetics.

MEPROBAMATE: Use lowest effective dose, particularly in elderly and/or debilitated, to preclude over-sedation. Meprobamate is metabolized in the liver and excreted by the kidney; to avoid excess accumulation exercise caution in its use in patients with compromised liver or kidney function. Meprobamate occasionally may precipitate seizures in epileptic patients. It should be prescribed cautiously and in small quantities to patients with suicidal tendencies.

ADVERSE REACTIONS: ASPIRIN: May cause epigastric discomfort, nausea, and vomiting. Hypersensitivity reactions, including urticaria, angioneurotic edema, purpura, asthma, and anaphylaxis may rarely occur. Patients receiving large doses of salicylates may develop tinnitus.

MEPROBAMATE: CNS: Drowsiness, ataxia, dizziness, slurred speech, headache, vertigo, weakness, paresthesias, impairment of visual accommodation, euphoria, overstimulation, paradoxical excitement, fast EEG activity.

GI: Nausea, vomiting, diarrhea.

CARDIOVASCULAR: Palpitation, tachycardia, various forms of arrhythmia, transient ECG changes, syncope, hypotensive crisis.

ALLERGIC OR IDIOSYNCRATIC: Milder reactions are characterized by itchy, urticarial, or erythematous maculopapular rash, generalized or confined to the groin. Other reactions include leukopenia, acute nonthrombocytopenic purpura, petechiae, ecchymases, eosinophilia, peripheral edema, adenopathy, fever, fixed drug eruption with cross-reaction to carisoprodal, and cross-sensitivity between meprobamate/mebutamate and meprobamate/carbromal. Rare, more severe hypersensitivity reactions include hyperpyrexia, chills, angioneurotic edema, bronchaspasm, aliguria, and anuria. Also, anaphylaxis, exfoliative dermatitis, stomatitis, and proctitis. Stevens-Johnson syndrome and bullous dermatitis have occurred.

HEMATOLOGIC (SEE ALSO "ALLERGIC OR IDIOSYNCRATIC"): Agranulocytosis, aplastic anemia have been reported, although no causal relationship has been established, and thrombocytopenic purpura.

OTHER: Exacerbation of porphyric symptoms.

DOSAGE AND ADMINISTRATION: Usual dose is one or two tablets, 3 to 4 times daily as needed for relief of pain when tension or anxiety is present. Not recommended for patients 12 years of age and under.

OVERDOSAGE: Treatment is essentially symptomatic and supportive. Any drug remaining in the stomach should be removed. Induction of vomiting or gastric lavage may be indicated. Activated charcoal may reduce absorption of both aspirin and meprobamate. Aspirin overdosage produces usual symptoms and signs of salicylate intoxication. Observation and treatment should include management of hyperthermia, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions. Suicidal attempts with meprobamate have resulted in drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse. Some suicidal attempts have been fatal. The following data, reported in the literature and from other sources, are not expected to correlate with each case (considering factors such as individual susceptibility and length of time from ingestion to treatment), but represent usual ranges reported. Acute simple overdose (meprobamate alone): Death has been reported with ingestion of as little as 12 gram meprobamate and survival with as much as 40 gram.

BLOOD LEVELS:

0.5-2.0 mg percent represents usual blood-level range after therapeutic doses. The level may occasionally be as high as 3.0 mg percent.

3-10 mg percent usually corresponds to findings of mild-to-moderate symptoms of overdosage, such as stupor or light coma.

10-20 mg percent usually corresponds to deeper coma, requiring more intensive treatment. Some fatalities occur.

At levels greater than 20 mg percent, more fatalities than survivals can be expected.

Acute combined overdose (meprobamate with other psychotropic drugs or alcohol): Since effects can be additive, history of ingestion of a low dose of meprobamate plus any of these compounds (or of a relatively low blood or tissue level) cannot be used as a prognostic indicator.

In cases of excessive doses, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Any drug remaining in stomach should be removed and symptomatic treatment given. Should respiration or blood pressure become compromised, respiratory assistance, CNS stimulants, and pressor agents should be administered cautiously as indicated. Diuresis, osmotic (mannitol) diuresis, peritoneal dialysis, and hemodialysis have been used successfully in removing both aspirin and meprobamate. Alkalinization of the urine increases excretion of salicylates. Careful monitoring of urinary output is necessary, and caution should be taken to avoid overhydration.

Relapse and death, after initial recovery, have been attributed to incomplete gastric emptying and delayed absorption.

HOW SUPPLIED: Bottles of 50 scored tablets.

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Beta Cell Function

(Continued from page 330)

function, and that maintaining a high degree of control is important to help preserve beta cell function.

In our opinion, many children with insulin-dependent diabetes receive delayed and inadequate treatment at the time of onset of symptoms. Too often precious time is lost by observing the response of the child to a modified dietary plan or a trial with oral hypoglycemic agents. The basic premise of our clinic, for children with diabetes, has been that the closer we can approximate a physiologic metabolic state, the greater will be the likelihood of normal growth and development and the lesser the likelihood of the development of both short and long-term complications.[24] The meals and snacks of children under our care are designed and structured to provide optimal nutrition, but are individualized so as to be in keeping with the eating habits of healthy, well-fed children of similar age and cultural background. It is well documented that, after diagnosis and treatment, most children with diabetes will have a good response for a variable period of time to an improved dietary program and a once daily injection of an intermediate type insulin.[3] We have found that at least two daily doses of intermediate acting insulin are desirable even during the remission period to provide a more physiologic repacement of insulin throughout the entire day, with peaks of activity at the time of the three daily meals. This insulin regimen also greatly lessens the possibility of hypoglycemic episodes and may prolong the remission. To prevent hypoglycemia, we also teach the parents and children how to vary the type and amount of food intake (especially in the snacks) to compensate for the wide daily fluctuation in physical activity of healthy children.[25]

To attain and maintain a high degree of control and prolong the remission period, we have found that hospitalization for a period of about three weeks is needed at the time of onset of the disease, not only to reestablish the nutritional status and determine maintenance insulin requirements but also to educate the parents and child. The combined efforts of a health team are needed to evaluate and prepare the family to continue the regimen after the child returns home.[25] Having an extended period of good health with a sense of well-being makes the child and the parents aware that the disease is controllable and usually makes them reluctant to accept lower degrees of control. The task is not easy. It requires understanding, judgment, and self-discipline but the effort is worthwhile, to maintain optimum health and decrease the odds against the insidious development of vascular changes.[24]

The availability of a reliable and sensitive C-peptide assay offers the opportunity of undertaking additional systematic animal and human studies to extend our knowledge of how to prevent destruction of, to promote regeneration of, and to preserve function of beta cells in insulin-dependent diabetic subjects.

Acknowledgements

The C-peptide determinations were done in the Diabetic Research and Training Center Laboratories of Arthur H. Rubenstein at the University of Chicago. The study was supported by NIH grants HL13632, AM13941 and AM20595, and by Eli Lilly Research Laboratories. W. Schondelmeyer and R. Hess provided technical assistance, and H. Waiches helped to abstract and record clinical and laboratory data for analysis.

References are available from the authors.

Drug Information

(Continued from page 346)

states no change.[4, 20] Methsuximide has been reported to increase phenytoin and phenobarbital serum levels.[22, 23]

Conclusions

Obviously, the potential drug interactions between the anticonvulsants are extensive, especially if three or more drugs are used. The complexity of these interactions is compounded by varying degrees of clinical significance.

The extent of enzyme induction varies between patients, and with the same patient depending on drug, dose, and length of therapy.[24] An enzyme inducer will have little or no effect on enzymes maximally induced. Alternatively, withdrawal of an enzyme inducer could have very significant effects. Induction effects on phenytoin are potentially more significant due to its dose dependent kinetics. A small change in metabolism can cause a disproportionate change in the serum levels of phenytoin.

An awareness of these potential drug interactions should be helpful to the clinician when modification of a patient's therapy is necessary.

Submitted by
Steven J. Schuller, R.Ph.

References are available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

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Percutaneous Transluminal Angioplasty

(Continued from page 334)

clinically in these patients. There may be a potential for redilatation or the need for arterial reconstructive surgery in some patients.

Percutaneous transluminal angioplasty is an effective treatment method when patients are properly selected. The early followup data is very encouraging. Continued evaluation of this procedure with objectively documented results using noninvasive Doppler studies is needed to determine its long term effectiveness and define its role in the treatment of vascular occlusive disease.

Summary

Percutaneous transluminal angioplasty was used to treat 22 patients with vascular occlusive disease of the lower extremities. Indications for angioplasty were claudication in 19 (86%) and rest pain in three (14%). The procedure was technically satisfactory in 21 (94%) and all (100%) showed initial clinical improvement. At mean followup of 9.4 months, 17 (85%) were either asymptomatic or improved, and three (15%) were failures. Late followup ankle-arm indices after exercise in 12 patients were improved when compared to pre-angioplasty levels; however, five had a decrease when compared to post-angioplasty levels. Late followup ankle-arm indices after exercise in ten patients were improved when compared to pre-angioplasty levels; however, five showed a decrease when compared to post-angioplasty levels. This study shows that angioplasty is effective in treating iliac and femoral artery stenoses in selected patients. Noninvasive arterial evaluation reflects the physiologic status of patients and may be a more sensitive indicator than clinical symptoms. Continued evaluation with objectively documented results is needed to define the role and determine the long term results of transluminal angioplasty.

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Theories of Dysmenorrhea

(Continued from page 340)

the etiology. They concluded that dysmenorrhea is significantly related to role acceptance. In other words, women who experience the most discomfort associated with menstruation are less likely to accept other aspects of a sexual role.[24]

However, in 1973, K. Jean and R. John Lennane found no valid basis for the attitude that dysmenorrhea is psychosomatic.[25]

The new concepts in dysmenorrhea are in biochemistry, physiology, and pharmacology. An important find is that prostaglandins are found in so many human tissues. When used to induce abortion, prostaglandins cause nausea and vomiting — two symptoms associated with dysmenorrhea. Thus, prostaglandin inhibitors are now being studied with promising results.

In 1975, Hansen and Secher postulated that the pain of essential dysmenorrhea may be due to a change in the tonus of the uterine musculature. They found that stimulating beta receptors in the myometrium successfully prevents premature labor, and used beta stimulators to treat dysmenorrhea with equivocal results.[26]

Oral contraceptives are also being used to treat dysmenorrhea since they prevent ovulation and anovulatory cycles are not painful.

Thus, it may be seen that although dysmenorrhea has been studied for many years, doctors are no closer to defining it or to finding a completely satisfactory treatment than they were in the time of Edward Clarke. There are now more drugs available to treat the symptoms, but the question still remains as to whether the symptoms are physiologic or psychosomatic.

The attitude of the physician to the problem today is as mixed as the theories of its etiology. Once again, women are pushing for increased freedom and may be perceived as a threat to some men who are happy with the status quo. Thus comments about the inferiority of women such as that by Edgar Berman are often heard. But they are not a part of "scientific" reports such as those seen in the 1870s. Although physicians may hold such beliefs in private, they do not include them in research reports

which must be based on fact.

The 19th century physician used his own observations to back what we now would regard as ludicrous studies. The problem with this past medical theorizing is that it has remained a part of the popular ideology of today. Thus, women are confronted with a much more subtle problem that is more difficult to dispute.

References and a bibliography of supplemental reading material are available from the author.

Malnutrition

(Continued from page 345)

Answers

1. c
2. d
3. c
4. e
5. d

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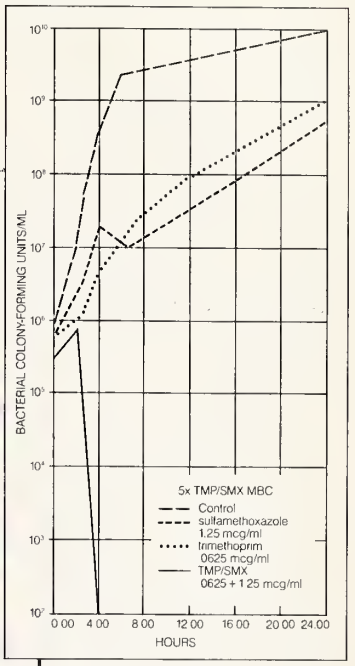
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Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.
For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.
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Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.
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Kill curve kinetics of Bactrim and its individual components against *E. coli in vitro*.[1]

Percent of isolates of common uropathogens sensitive to BACTRIM and to other antimicrobials

	Escherichia coli	*Klebsiella pneumoniae*	*Proteus mirabilis*	*Proteus vulgaris*	*Proteus sp.*	*Enterobacter sp.*	*Enterobacter aerogenes*	*Enterobacter cloacae*
BACTRIM	**96%** 328,598	**89%** 63,551	**93%** 62,691	**84%** 4174	**91%** 4381	**88%** 1736	**96%** 7326	**92%** 11,856
ampicillin	72% 345,209	5% 65,466	85% 67,338	16% 4381	70% 4402	10% 1775	9% 7759	11% 12,091
cephalexin†	81% 343,339	85% 67,031	92% 67,027	16% 4398	80% 4365	22% 1834	13% 7571	12% 11,846
nitrofur-antoin	97% 307,785	65% 61,162	7% 59,775	13% 3848	14% 3059	67% 1707	60% 7109	67% 10,830

†Analogous to cephalothin, the primary antibiotic disc used in testing.
Source: The Bacteriologic Report, BAC-DATA Medical Information Systems, Inc., Winter Series, 1981-82.
Numbers under percentages refer to the projected number of isolates tested.

The bactericidal action of Bactrim has been demonstrated *in vitro* on laboratory strains of *E. coli*[1,2] and on clinical isolates of *E. coli, Klebsiella-Enterobacter, Proteus mirabilis* and *Morganella morganii*[3]—the most common causative organisms of urinary tract infections.[4] More than 100 published studies attest to the efficacy of Bactrim in recurrent urinary tract infections due to these organisms.[5] In comparative studies with other antimicrobials, Bactrim has consistently demonstrated unsurpassed efficacy during therapy.[6-11]

Resistance to Bactrim develops more slowly than to either of its components alone *in vitro*.* Among urinary tract isolates, resistance has rarely emerged in susceptible strains.[5,12] Bactrim is contraindicated in pregnancy at term, during lactation, in infants less than two months old and in documented megaloblastic anemia due to folate deficiency.

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Arterial Trauma

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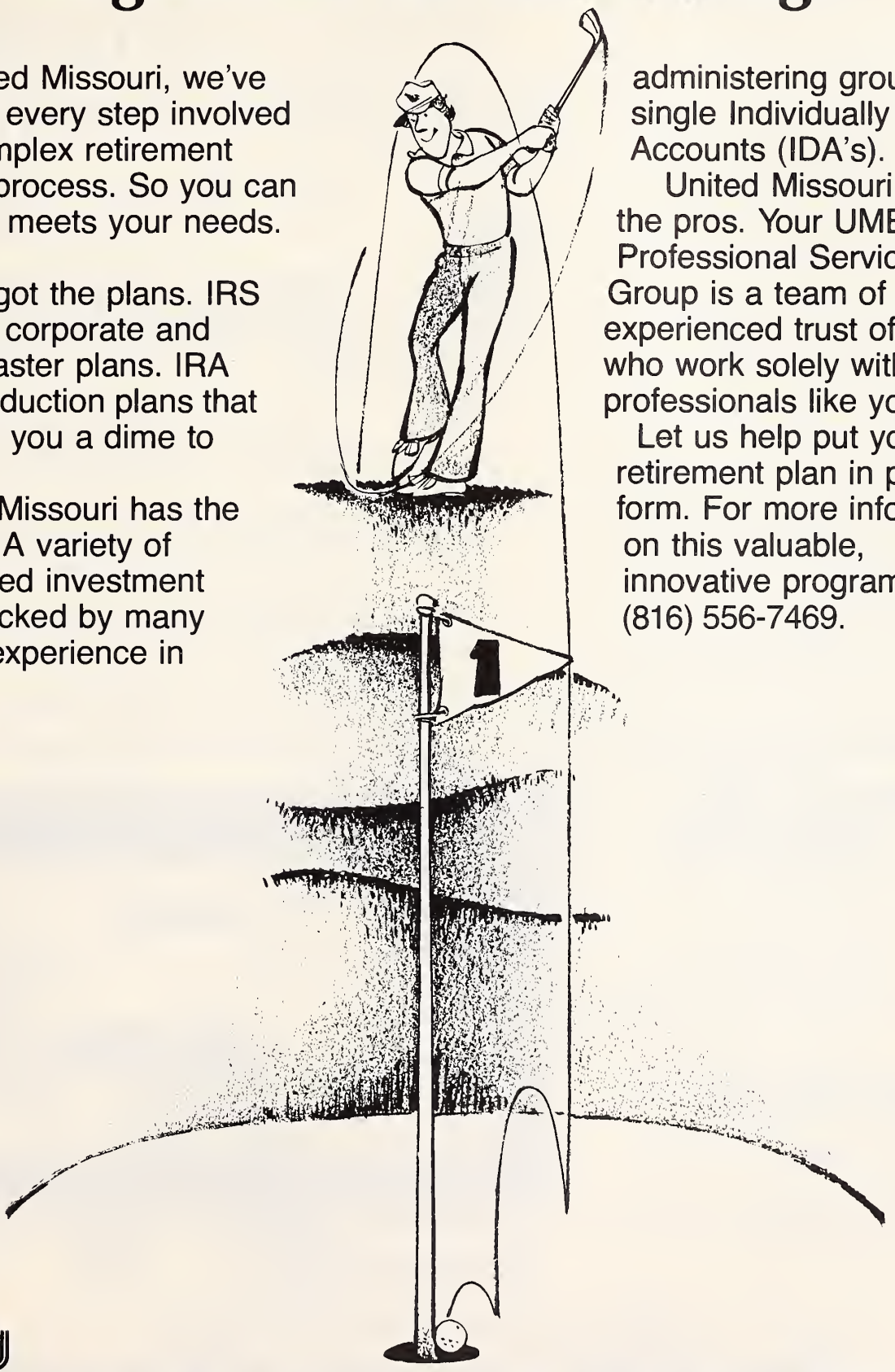
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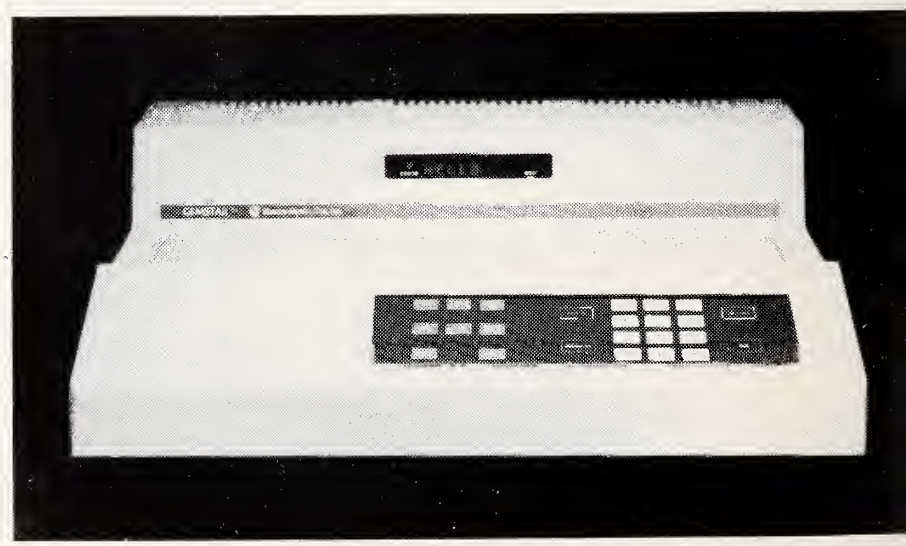
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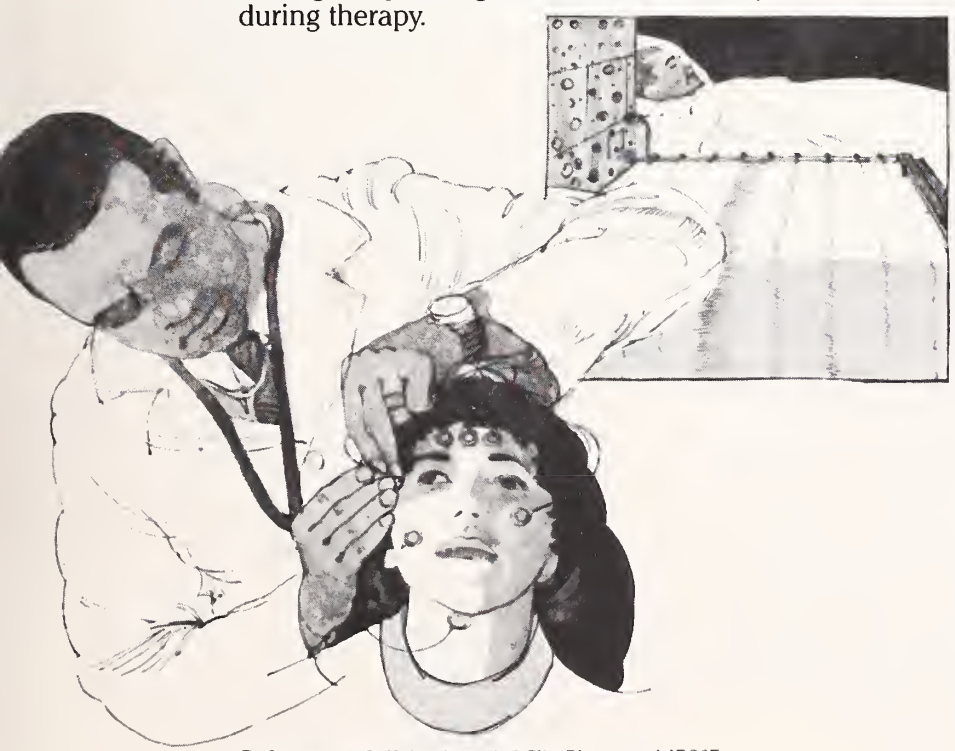
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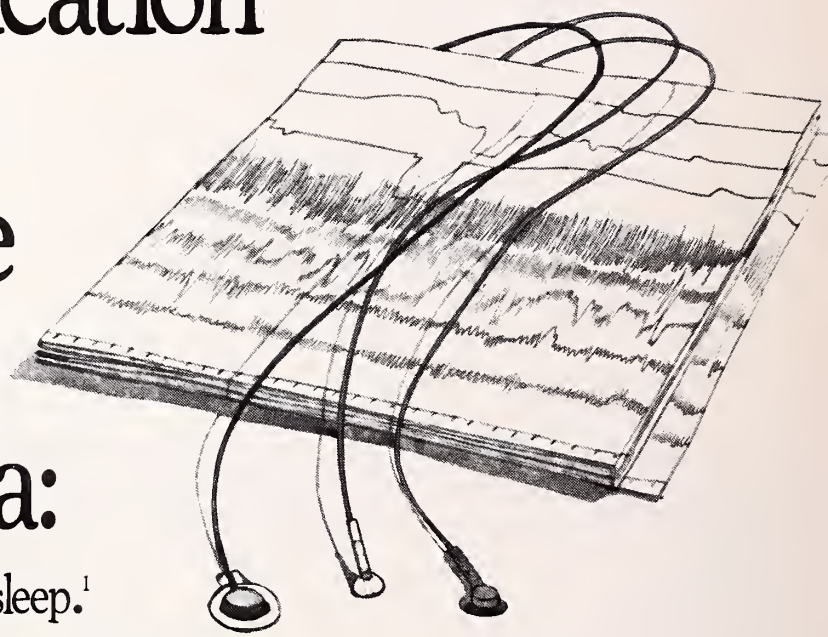
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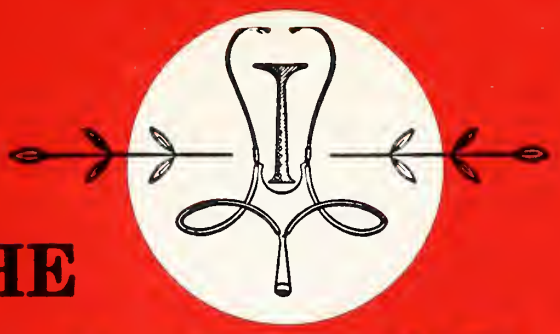
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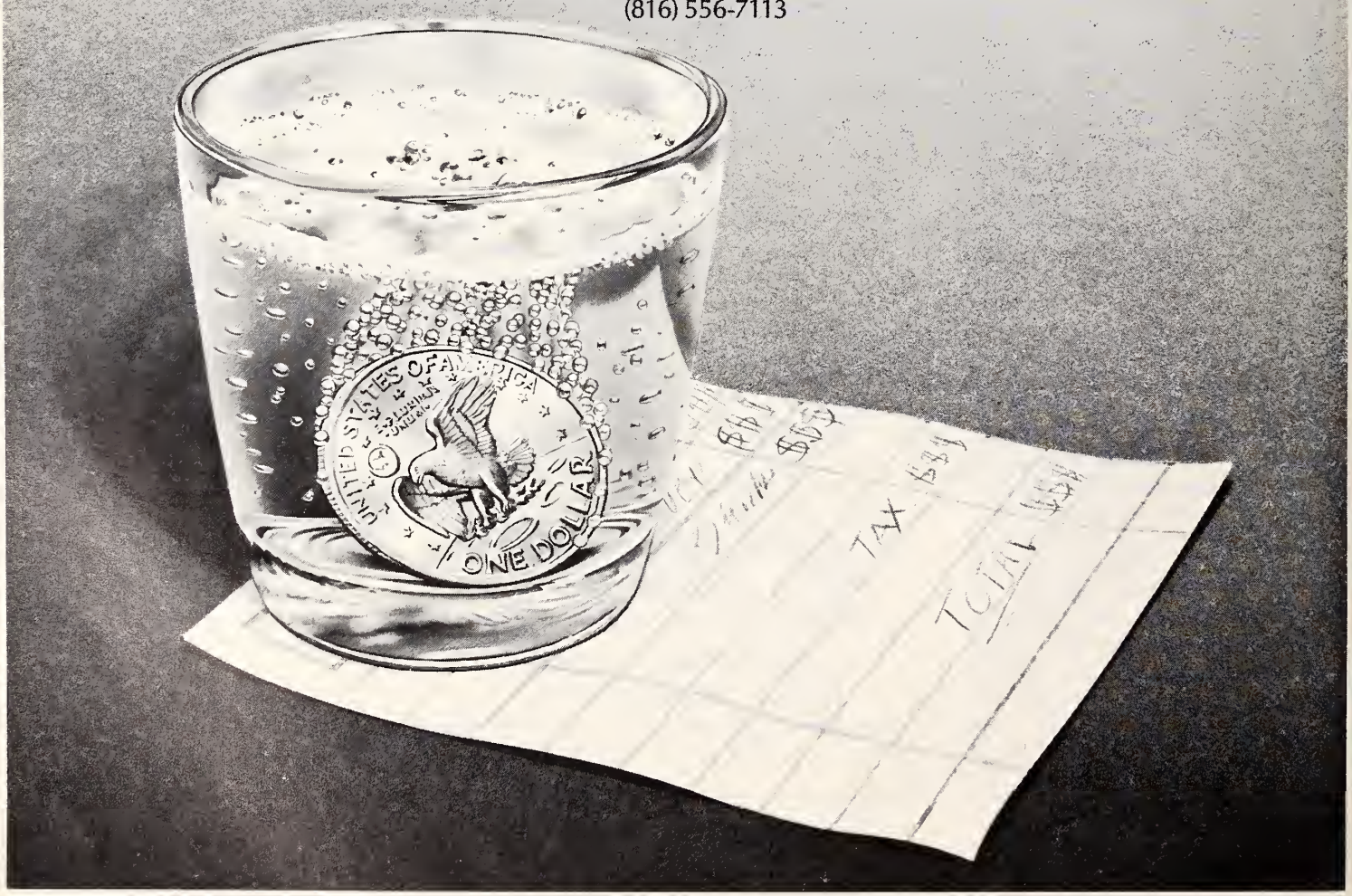
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Official Proceedings

1983 Annual Meeting of the House of Delegates

Transactions of the 124th Annual Session of the Kansas Medical Society are published in this issue of *The Journal*.

The Resolutions are printed in numerical order under the Minutes of the Second House of Delegates Session. Those Resolutions that were not adopted but were referred for further study or information are so indicated. The Resolutions that failed to pass are retained in the Official Minutes at the Executive Office, but are not reported here.

FIRST SESSION

The First Session of the House of Delegates of the Kansas Medical Society met on Friday, May 6, 1983, beginning at 9:00 AM at the Holiday Inn Holidome, Lawrence. The meeting was called to order by G. Rex Stone, M.D., Speaker. Dr. Stone briefly reviewed the order of business for the day and explained that the House was composed of elected officers, Past Presidents, representatives of specialty societies and the duly elected delegates from the component medical societies. He stated that the House would follow the *Sturgis Standard Code of Parliamentary Procedure*. Dr. Stone then invited Kermit G. Wedel, M.D., President of the Kansas Medical Society to assume the podium.

Dr. Wedel welcomed the Delegates and guests to the Annual Meeting of the Kansas Medical Society and extended greetings from the KMS officers. He then introduced Joseph F. Boyle, M.D., Chairman of the AMA Board of Trustees.

Dr. Boyle reviewed many of the AMA activities at the federal level, including recent changes in Medicare and Medicaid. He commended Dr. Wedel for his leadership and bold changes in direction at the state level, and indicated that now is the time for commitment and leadership.

After announcing the presence of the official quorum, the Speaker appointed the following tellers: Warren E. Meyer, M.D., Wichita, Chairman; David A. Leitch, M.D., Garnett, and Thomas F. Taylor, M.D., Salina. Ballots for the primary election were distributed and the Delegates voted for the following slate:

PRESIDENT ELECT: F. Calvin Bigler, M.D., Garden City

FIRST VICE PRESIDENT: Clair C. Conard, M.D., Dodge City

SECOND VICE PRESIDENT: Franklin G. Bichlmeier, M.D., Kansas City; David H. Clark, M.D., Salina; and Ivan E. Rhodes, M.D., Wichita

CONSTITUTIONAL SECRETARY: K. William Bruner, Jr., M.D., Wichita

TREASURER: Richard Meidinger, M.D., Topeka; and Wayne O. Wallace, Jr., M.D., Atchison

AMA DELEGATES: Lew W. Purinton, M.D., Wichita; and Kermit G. Wedel, M.D., Minneapolis

AMA ALTERNATE DELEGATES: Ray E. Allen, M.D., Liberal; Jimmie A. Gleason, M.D., Topeka; Robert P. Hudson, M.D., Olathe; and Linda D. Warren, M.D., Hanover

SPEAKER: G. Rex Stone, M.D., Manhattan

VICE SPEAKER: Franklin G. Bichlmeier, M.D., Kansas City

David E. Gray, M.D., presented the Necrology Report, after which the House stood for a moment of silence in tribute to the members who departed during the past year.

Dr. Gray then presented the report of the Editorial Board, after which he and Dr. Wedel exchanged bound volumes of *The Journal*.

There followed reports by Dr. Wedel, President; Steve Carter, Executive Director; and Dr. Gleason, Chairman of the Legislative Committee. Dr. Gleason also announced that Erwin T. Janssen, M.D., Topeka, will assume the Chairmanship of the Legislative Committee for the coming year.

The Speaker announced the Primary Election results for the office of Second Vice President. Retained on the ballot were Drs. Bichlmeier and Rhodes. After a brief address by Meldon Laury (David), KMS Auxiliary President, Dr. Stone directed the Delegates' attention to the Resolutions in their packets. All such Resolutions are considered automatically introduced. New business introduced from the floor pertained to a Resolution to commend Executive Vice Chancellor Waxman and one on KaMPAC. Dr. Stone then introduced the Reference Committee: John W. Young, M.D., Shawnee Mission; Tell B. Copening, M.D., Iola; Edwin D. Rathbun, M.D., Liberal; David E. Street, M.D., Wichita; and Wayne O. Wallace, Jr., M.D., Atchison. He announced that the Reference Committee will meet in this same room at 1:30 PM and invited all those interested to help the Committee deliberate the issues.

Dr. Stone announced the following six Council Districts who needed to elect Councilors: numbers

6, 7, 10, 12, 16, and 18.

After briefly reviewing the format of the Convention for the balance of the day, the Speaker adjourned the First Session of the House of Delegates at 11:00 AM.

Necrology Committee—
David E. Gray, M.D., Chairman

The death of a physician has been celebrated in literature as being of a special quality. Physicians themselves, constantly confronting death somewhere between physiologic fact and philosophic dilemma, are aware that for them it is no different since it brings the one certainty of ultimate equality. For the physician no less than the lay person, it can come with shocking prematurity or over-long delay. The roster of members of the Kansas Medical Society whose deaths have come to the attention of your Committee since our last meeting encompasses the full range and includes:

Name and address	*Age*	*Date*
Myrtle S. Carney, M.D., Fort Worth, Texas	95	Unknown
G. Ralph Combs, M.D., Leavenworth	104	1/17/83
Frank A. Dlabal, M.D., Wilson	75	7/11/82
Frank A. Emery, M.D., Wichita	77	4/22/82
Charles G. Foster, M.D., Topeka	62	4/1/83
Robert C. Goering, M.D., Wichita	56	4/16/82
Rene M. Gouldner, M.D., Wichita	94	3/24/83
Peter E. Hiebert, M.D., Shawnee Mission	79	2/2/83
Pacharee Hompluem, M.D., Parsons	39	5/5/82
Howard U. Kennedy, M.D., Topeka	64	11/14/82
Richard S. McKee, M.D., Leavenworth	76	1/12/82
Robert W. Myers, M.D., Newton	71	3/26/83
Curtis A. Nystrom, M.D., Topeka	59	2/12/83
Glen R. Peters, M.D., Kansas City	70	2/3/83
Arthur L. Pincomb, M.D., Olathe	62	1/3/83
Henry L. Regier, M.D., Kansas City	101	10/?/82
Francis H. Relihan, M.D., Smith Center	97	3/10/82
Frank A. Rieke, M.D., Kansas City	66	2/23/83
Clemens Rucker, M.D., Topeka	95	5/21/82
J. Colbert Simpson, M.D., Salina	74	9/19/82
Jack T. Stewart, M.D., Wichita	51	4/18/82
Elmer G. Stolz, M.D., Wichita	64	10/25/81
Peter K. Wiens, M.D., Ness City	58	1/9/83
Vernon C. Wiksten, M.D., Topeka	70	1/19/82

Editorial Board—
David E. Gray, M.D., Chairman

You may have noticed that this session has a somewhat different look from its predecessors. Ordinarily there is a line-up of committee chairmen reminiscent of the annual parade of tenants coming to pay their rents, tug their forelocks, and declare their fealty to the lord of the manor. This year's lord, in an exercise of parliamentary cost containment, has admitted only a select few to the front parlor, all

References:
1. Stone PH, Turi ZG, Muller JE: Efficacy of nifedipine therapy for refractory angina pectoris. *Am Heart J* 104:672-681, September 1982.
2. Antman E, Muller J, Goldberg S, et al: Nifedipine therapy for coronary-artery spasm: Experience in 127 patients. *N Engl J Med* 302:1269-1273, June 5, 1980.

BRIEF SUMMARY
PROCARDIA® (nifedipine) **CAPSULES** For Oral Use

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents: (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

More detailed professional information available on request.

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Quotes from an unsolicited letter received by Pfizer from an angina patient. While this patient's experience is representative of many unsolicited comments received, not all patients will respond to Procardia nor will they all respond to the same degree.

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"I shop, cook and can plant flowers again."

"I have been able to do volunteer work...and feel needed and useful once again."

PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks,[1] taking fewer nitroglycerin tablets,[2] doing more, and being more productive once again.

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%).



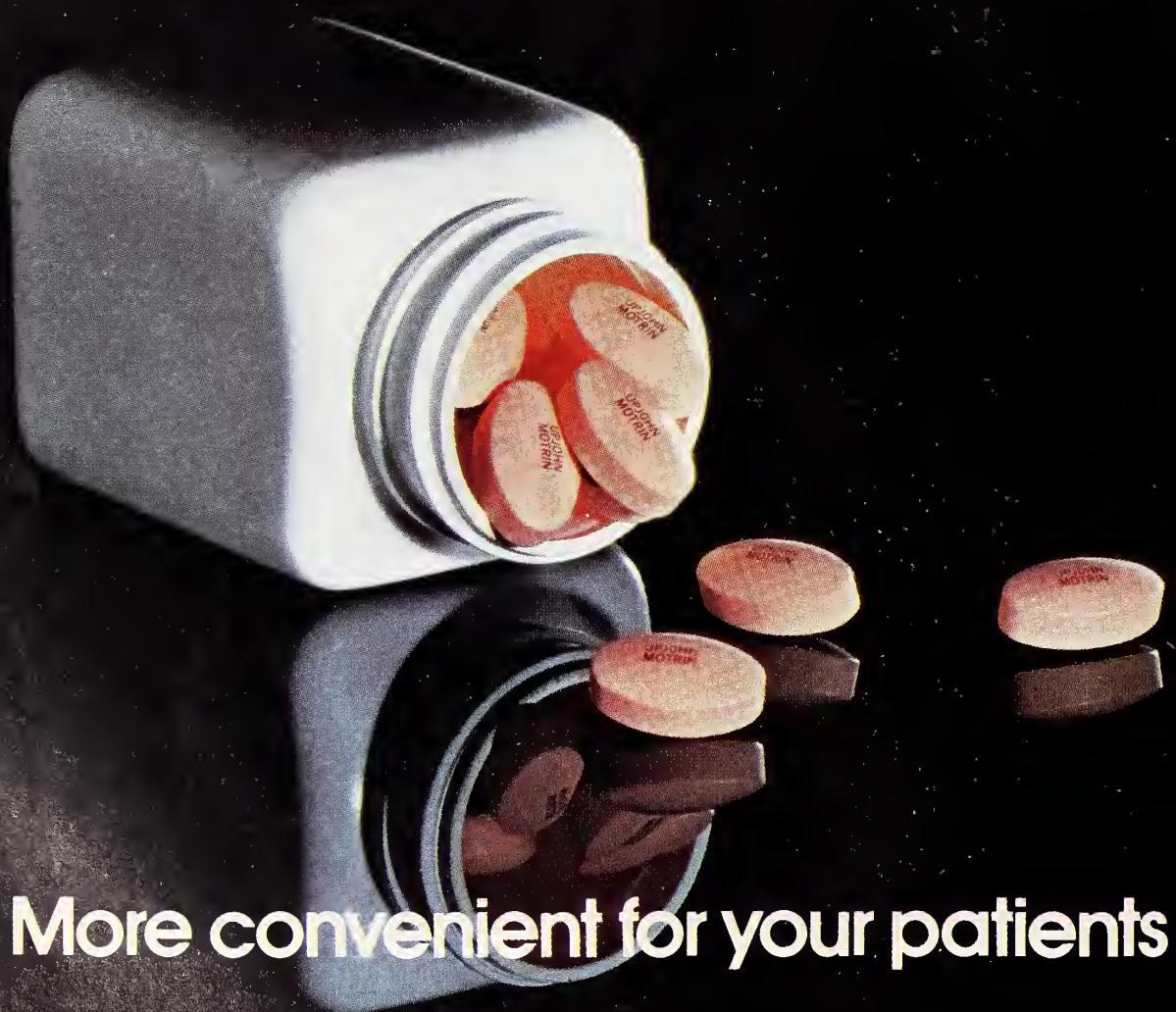
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J-9043-4 July 1981

understandable on a political basis except one — the Editorial Board. For fear this may have been a mistake, I shall therefore try to keep a low profile and hold to the brevity that has been the one redeeming feature of my previous reports — not to mention the time saved since there is no forelock to tug.

The year has been a reasonably tranquil one for the *Journal* but one change of note has occurred. With his retirement, Jesse Rising has terminated a long and valued service as the Editorial Board member representing the Medical Center in Kansas City. His primary task has been to ride herd on his colleagues there to develop the annual Medical Center issue. His success over the years is a matter of record, and the *Journal* is grateful to him for that and for his other services. A logical and happy choice for his successor was Jack Walker — logical since his status and reputation assured us of a smooth transition and continued high quality of purpose, happy for the Board as it welcomes a personable, familiar, and valued addition. Anyone who needs further introduction to the gentleman has no business here anyway.

As evidence that the *Journal* can be counted on for constancy in a tumultuous world, we have just celebrated an anniversary of some note. For the past thirty years, the *Journal* has been published by the Ovid Bell Press, Inc. of Fulton, Missouri. It all began in the days of Orville Clark's editorship with Oliver Ebel managing our business affairs and Pauline Farrell doing the real work. The Ovid Bell Press, Inc., stables a variety of periodicals including some ten or more medical journals and we obviously feel it has been a mutually rewarding relationship. As with a marriage of thirty years, the mysteries and passions of earlier times have progressed to a satisfying sense of comfort and belonging which seems destined to continue.

You may have noted that the cover of the *Journal* has taken on a more vivid hue. While this is a change of less than earth-shaking character, it is quite remarkable for a publication as staid as the *Journal* and we hope, like someone sporting a fancy exercise suit, that observers will interpret it as indication of the robust substance of the contents.

Our new business manager, Steve Carter, has manfully adapted to the peculiarities of the Board's laid-back approach to the facts of publication life. It can be reported that a little fancy bookkeeping footwork has brought the *Journal* under the administration's financial umbrella and to the best of my knowledge we are still solvent since it is not likely they would have let us in if we weren't. I am perfectly content to inform you that if it is ever otherwise, it

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will be up to Steve to confront you with the news. Steve has assumed his role in the *Journal* management with tactful interest and discerning interpretations although I suspect that, in common with many others, he's still not quite sure what to make of the Editor.

It seems totally inadequate simply to say "Thank you" to Val and Eleanor for their contributions to the *Journal* and constant and patient assistance to the Editor, but sometimes the simpler statement is more emphatic than the prolonged recital of praise with the hollowness of excess. I hope they accept the brevity of this note as indication that this is one of those occasions. If you have had reason to contact the *Journal* office, you should know that any prompt and effective response originated with one of them. If you have heard nothing, you should know that the matter has been referred to the Editor whose file of unfinished business is equalled only by that of the Congress.

To return to the change of format of this meeting, I do want to share with you my private theory as to why the Editorial Board was accorded the privilege of coming before you. I believe it stemmed from the President's fear that if he cut me out, he wouldn't receive his bound volume of the *Journal* which is, after all, the prime reason anyone subjects himself to the masochistic exercise of trying to run this outfit. So, to relieve Kermit's anxiety, it is my pleasant duty to offer the Board's testimony to that ancient aphorism, "When you care enough to send the very best."

SECOND SESSION

The Second Session of the House of Delegates was called to order by the Speaker, G. Rex Stone, M.D., on Sunday, May 8, 1983, at 8:00 AM at the Holiday Inn Holidome, Lawrence.

The Speaker placed some rules before the body and announced that *Sturgis Standard Code of Parliamentary Procedure* would be followed at this meeting. Each delegate was to be allowed an opportunity to be heard on every question, but except for the person who makes the motion, all delegates were asked to cooperate by being heard only once upon a single question. He also announced that a quorum was present.

The Speaker appointed Drs. David A. Leitch,

(Continued on page 416)

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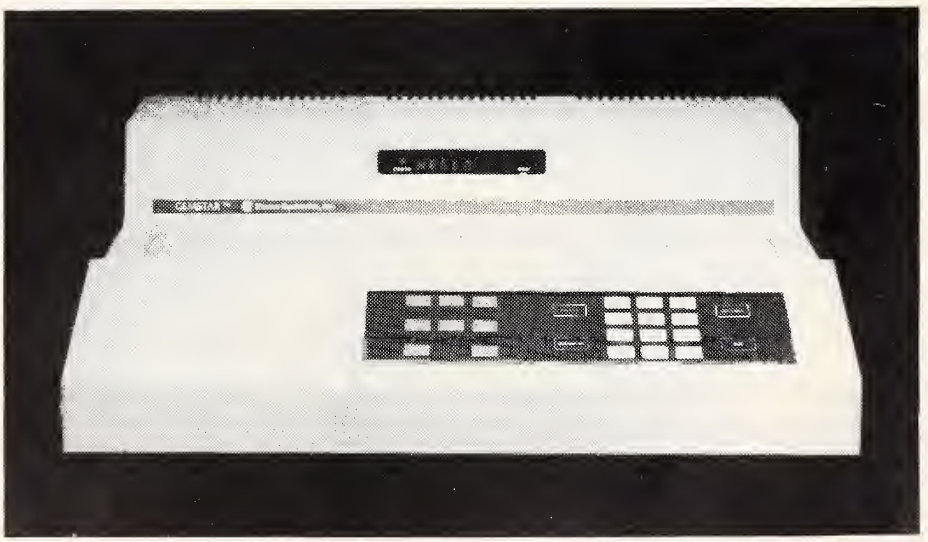
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AUXILIARY NEWS

An Open Letter to Kansas Physicians

Dear Doctor:

Diane Sanders (Alan), the new President of KMSA, asked that I send a report to you concerning our Convention in Lawrence, May 5-7, 1983.

Accompanied by a cheery welcome from Douglas County Medical Auxiliary Convention members wearing beautiful prairie aprons displaying a wheat motif — 1983 convention logo — registration took place in the lobby of the new and beautiful Lawrence Holidome.

A surprise was in store for those who attended Sports Day in the form of an announcement by K.U. Chancellor Gene Budig in his welcoming message that D. Kay Clawson, M.D., had been chosen as the new Executive Vice-Chancellor in charge of the K.U. Medical School.

Meldon Laury (David) of Ottawa, President, called the Pre-convention Board of Directors meeting to order, and the 58th Annual Convention of the KMSA was under way. A great deal of this meeting time was spent discussing By-laws and Standing Rules, and how to make them conform to the AMAA and to the times. There was also a tour of the Helen Foresman Spencer Museum of Art at Kansas University and a luncheon honoring past state presidents.

Presiding at the luncheon and General Session I with her usual warmth and enthusiasm, Meldon Laury brought charm to the occasion. The highlight of the afternoon — the keynote address — was presented by honored guest Betty Payne (Torrence), Newburgh, New York, AMAA President. Mrs. Payne endeared herself to us immediately with her complimentary comments about the middle west portion of this country and her devotion to the United States — her adopted land. Her message concerned our need to change from looking to the government for fulfillment of needs to using our volunteer resources for accomplishment. She pointed out that "our organization represents aids to change from the 'me decade' to the 'we decade.' " She reminded us of our need for fellowship — never to be forgotten — and asked us to accept the present time as one of challenge to be committed to volunteerism.

The evening dinner honoring Mrs. Payne, AMAA President, and Dr. Kermit Wedel, KMS President, and Mrs. Wedel, was attended by 220 guests, many in prairie attire.

The events of the evening came very close to being upstaged by a tornado that took one life and damaged 100 homes in Topeka, just a few miles west. Holidome employees treated the situation with expertise — ushering us into inner hallways, where we spent approximately 45 minutes, and serving the hot cornish hen immediately upon our return to the table. Did food ever taste better? Community spirit blossomed into the joy of living! The following auction was a tremendous success — thanks to Phoebe Godwin (Philip) and her committee; Mr. Loren Freeman, a professional auctioneer who donated his services; and all of those who donated items and purchased them so generously. The Virginia Reel followed closely on the heels of the auction, and dancing continued until the wee hours.

Saturday was another full day.

—We learned that Friday evening's auction, together with boutique sales, had earned $5,814 for AMAERF and $375 (from the sale of gold-plated stethoscopes) to help defray convention expenses.

—Carol Loeffler (James), AMAERF Chairman, reported $20,966.33 raised this year for this project.

—Kermit G. Wedel, M.D., KMS President, greeted us, complimented us on our meeting, and humorously suggested we meet in Lawrence annually.

—Roger D. Warren, M.D., KaMPAC Chairman, told us that our freedoms are being assailed be-

cause of changing attitudes — much of it political. New limitation for contributions to political candidates, plus the fact that voluntary donations are coming in from only about 10 per cent of our membership, makes KaMPAC's tasks virtually impossible under the present system. We need to consider alternatives.

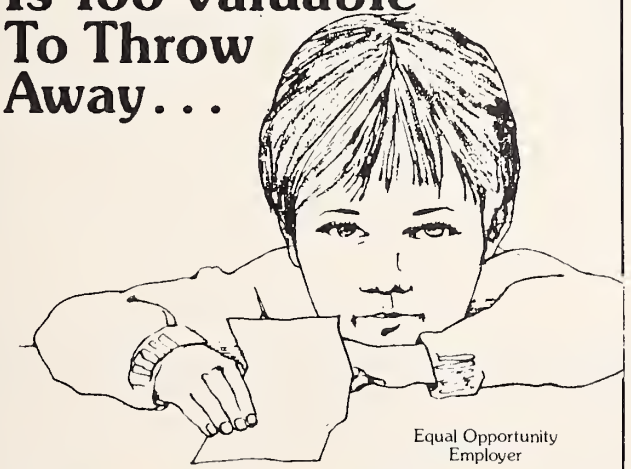
—Steve Carter, KMS Executive Director, warned us that, because of changes by the federal government, all practice in Kansas will change drastically within the next few years. He stated that Dr. Kermit Wedel spent half of his time last year dealing with legislation on a state and national basis. Mr. Carter stated that the government will use hospitals to control the practice of medicine — there will be 30 per cent too many physicians by 1990 — clinics will spring up everywhere — our health insurance will never be the same — and from 10 to 50 hospitals will very probably close in Kansas in the next few years. His prescription for assistance to the ailing profession of our spouses is to maintain better communication with patients, who are still our strong advocates.

The luncheon at the Lawrence Country Club was highlighted by the greeting from Mrs. Payne; the installation of officers with many members of their families present; Diane Sander's address; a very delightful and colorful performance by the Scandinavian Dancers — ranging in age from grandchild to grandmother — and including Faith Whitley (Douglas), President of the Johnson County Auxiliary; and of course Meldon Laury's wonderful personality pulling it all together, her "Potpourri of Talents" having finished a job. Well done!

The Post-convention Board of Directors meeting adopted a motion to develop with the Kansas Medical Society the Senior Citizens Forum across the state. The Treasurer reported $13,748.40 in total assets. The Summer Board Meeting will be held July 13-14 at the Lawrence Holidome.

Shirley Wilcox (Howard)
Publicity Chairman, Convention Committee
Douglas County Medical Auxiliary

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—G. Boss, MD et al

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"... (1) patients with extensive tophaceous disease...; (2) patients with a history of renal calculi... since a uricosuric drug may exacerbate renal stone disease; and (3) patients with significant renal disease... who are unlikely to respond to a uricosuric drug."[2]

—Edward W. Holmes, Jr, MD

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INDICATIONS AND USE: This is not an innocuous drug and strict attention should be given to the indications for its use. Pending further investigation, its use in other hyperuricemic states is not indicated at this time.
Zyloprim® (allopurinol) is intended for:
1. treatment of gout, either primary, or secondary to the hyperuricemia associated with blood dyscrasias and their therapy;
2. treatment of primary or secondary uric acid nephropathy, with or without accompanying symptoms of gout;
3. treatment of patients with recurrent uric acid stone formation;
4. prophylactic treatment to prevent tissue urate deposition, renal calculi, or uric acid nephropathy in patients with leukemias, lymphomas and malignancies who are receiving cancer chemotherapy with its resultant elevating effect on serum uric acid levels.

CONTRAINDICATIONS: Use in children with the exception of those with hyperuricemia secondary to malignancy. The drug should not be employed in nursing mothers.
Patients who have developed a severe reaction to Zyloprim should not be restarted on the drug.
WARNINGS: ZYLOPRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION.
In some instances a skin rash may be followed by more severe hypersensitivity reactions such as exfoliative, urticarial and purpuric lesions as well as Stevens-Johnson syndrome (erythema multiforme) and very rarely a generalized vasculitis which may lead to irreversible hepatotoxicity and death.
A few cases of reversible clinical hepatotoxicity have been noted and in some patients asymptomatic rises in serum alkaline phosphatase or serum transaminase have been observed. Accordingly, periodic liver function tests should be performed during the early stages of therapy, particularly in patients with pre-existing liver disease.
Patients should be alerted to the need for due precautions when engaging in activities where alertness is mandatory.
Occasional cases of hypersensitivity have been reported in patients with renal compromise receiving thiazides and Zyloprim concurrently. For this reason, in this clinical setting, such combination should be administered with caution.
In patients receiving Purinethol® (mercaptopurine) or Imuran® (azathioprine), the concomitant administration of 300-600 mg of Zyloprim per day will require a reduction in dose to approximately one-third to one-fourth of the usual dose of mercaptopurine or azathioprine. Subsequent adjustment of doses of Purinethol or Imuran should be made on the basis of therapeutic response and any toxic effects.
Usage in Pregnancy and Women of Childbearing Age:
Zyloprim should be used in pregnant women or women of childbearing age only if the potential benefits to the patient are weighed against the possible risk to the fetus.
PRECAUTIONS: Some investigators have reported an increase in acute attacks of gout during the early stages of allopurinol administration, even when normal or subnormal serum uric acid levels have been attained.
It has been reported that allopurinol prolongs the half-life of the anticoagulant, dicumarol. This interaction should be kept in mind when allopurinol is given to patients already on anticoagulant therapy, and the coagulation time should be reassessed.
A fluid intake sufficient to yield a daily urinary output of at least 2 liters and the maintenance of a neutral or, preferably, slightly alkaline urine are desirable to (1) avoid the theoretic possibility of formation of xanthine calculi under the influence of Zyloprim therapy and (2) help prevent renal precipitation of urates in patients receiving concomitant uricosuric agents.
Patients with impaired renal function require less drug and should be carefully

"The most important therapeutic measure is the administration of a drug which will block urate synthesis. The agent available at present is allopurinol (Zyloprim . . .) which is very effective and of low toxicity."[3]

—Alfred Jay Bollet, MD

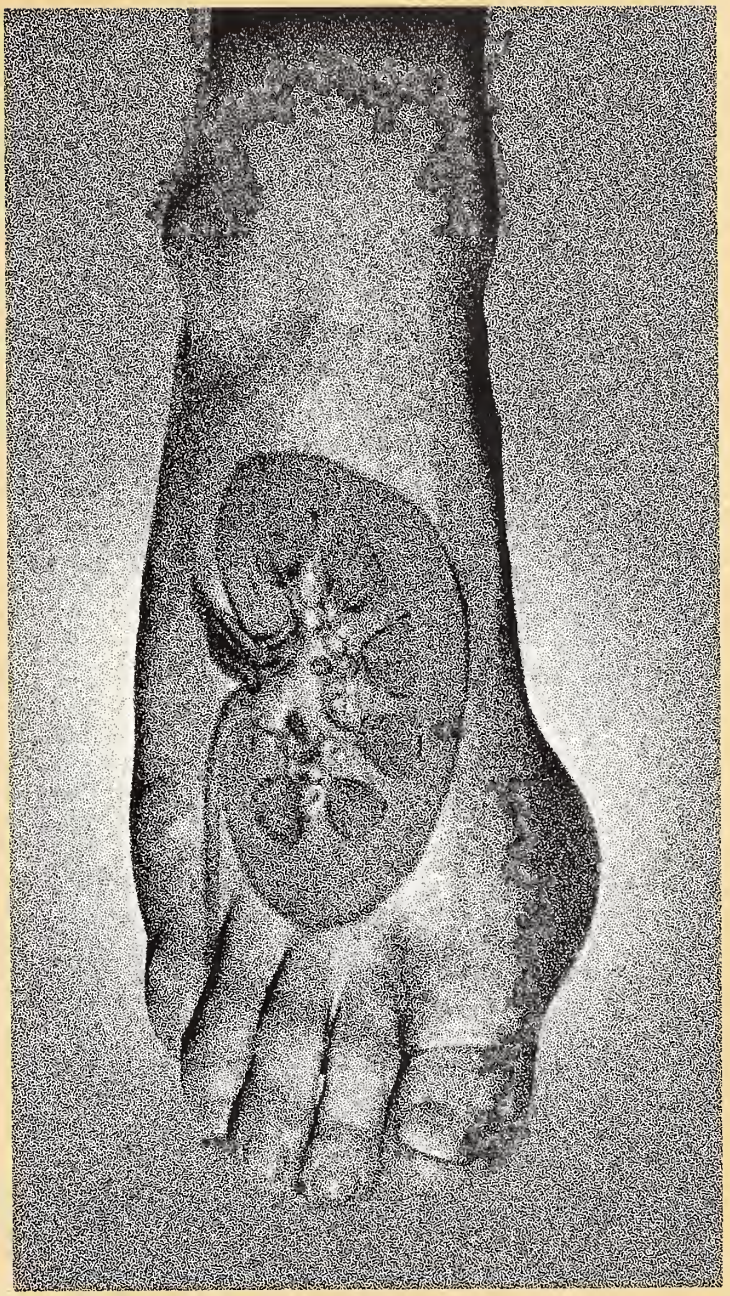
". . . allopurinol treatment appears to retard the progression of renal dysfunction."[4]

—T. Gibson, MD et al

LOW INCIDENCE OF TOXICITY

"Clinical experience with allopurinol suggests that most patients tolerate this drug well—a finding strongly supported by our data. Undesired or unintended effects of therapy were reported in only 1.8% of 1835 consecutive recipients."[5]

—G. T. McInnes, MD



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observed during the early stages of Zyloprim administration and the drug withdrawn if increased abnormalities in renal function appear.

In patients with severely impaired renal function, or decreased urate clearance, the half-life or oxipurinol in the plasma is greatly prolonged. Therefore, a dose of 100 mg per day or 300 mg twice a week, or perhaps less, may be sufficient to maintain adequate xanthine oxidase inhibition to reduce serum urate levels. Such patients should be treated with the lowest effective dose, in order to minimize side effects.

Mild reticulocytosis has appeared in some patients.

Periodic determination of liver and kidney function and complete blood counts should be performed especially during the first few months of therapy.

ADVERSE REACTIONS:

Dermatologic: Because in some instances skin rash has been followed by severe hypersensitivity reactions, it is recommended that therapy be discontinued at the first sign of rash or other adverse reaction (see WARNINGS). Skin rash, usually maculopapular, is the adverse reaction most commonly reported. The incidence of skin rash may be increased in the presence of renal disorders.

Exfoliative, urticarial and purpuric lesions, Stevens-Johnson syndrome (erythema multiforme) and toxic epidermal necrolysis have also been reported.

A few cases of alopecia with and without accompanying dermatitis have been reported.

In some patients with a rash, restarting Zyloprim (allopurinol) therapy at lower doses has been accomplished without untoward incident.

Gastrointestinal: Nausea, vomiting, diarrhea, and intermittent abdominal pain have been reported.

Hepatic: Rare cases of granulomatous hepatitis and hepatic necrosis have been reported.

Vascular: There have been rare instances of a generalized hypersensitivity vasculitis or necrotizing angiitis which have led to irreversible hepatotoxicity and death.

Hematopoietic: Agranulocytosis, anemia, aplastic anemia, bone marrow depression, leukopenia, pancytopenia and thrombocytopenia have been reported in patients, most of whom received concomitant drugs with potential for causing these reactions. Zyloprim has been neither implicated nor excluded as a cause of these reactions.

Renal: Rare cases of renal failure have been reported in hypertensive patients who received thiazides and Zyloprim concurrently. Some patients had evidence of hypersensitivity to allopurinol.

Neurologic: There have been a few reports of peripheral neuritis occurring while patients were taking Zyloprim. Drowsiness has also been reported in a few patients.

Ophthalmic: There have been a few reports of cataracts found in patients receiving Zyloprim. It is not known if the cataracts predated the Zyloprim therapy. "Toxic" cataracts were reported in one patient who also received an anti-inflammatory agent; again, the time of onset is unknown. In a group of patients followed by Gutman and Yü for up to five years on Zyloprim therapy, no evidence of ophthalmologic effect attributable to Zyloprim was reported.

Drug Idiosyncrasy: Symptoms suggestive of drug idiosyncrasy have been reported in a few patients. This was characterized by fever, chills, leukopenia or leukocytosis, eosinophilia, arthralgias, skin rash, pruritus, nausea and vomiting.

OVERDOSAGE: Massive overdosing, or acute poisoning, by Zyloprim has not been reported.

HOW SUPPLIED: 100 mg (white) scored tablets, bottles of 100 and 1000; 300 mg (peach) scored tablets, bottles of 30, 100 and 500. Unit dose packs for each strength also available.

Complete information available from your local B. W. Co. Representative or from Professional Services Department PML.

U.S. Patent No. 3,624,205 (Use Patent)

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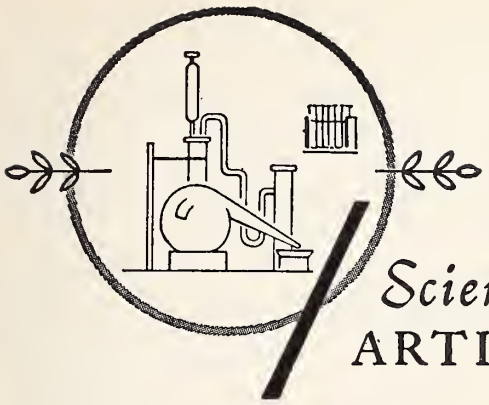
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Rabies in Kansas

Epidemiology and Treatment

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and **CPT ROBERT B. MOELLER, Jr., VC‡** ***Fort Detrick, Maryland***

Rabies, an acute viral illness, is almost uniformly fatal to humans. It most often results from the bite of a rabid animal. A decision to treat should be based on multiple criteria. A new prophylactic antirabies treatment regimen has been shown to be effective in preventing the exposed patient from developing rabies. Information concerning the shipment of animal remains for rabies testing is outlined, and a listing of Kansas County Health Department telephone numbers provides access to additional rabies information.

Rabies, once a scourge of mankind, is usually thought of as a lethal viral infection found only in developing countries. However, a rebirth of this dreaded disease may be near at hand in Kansas. Of great concern in the control of this disease is the increasing number of rabies positive wild animals isolated during the last five years in the United States.[1] The Centers for Disease Control (CDC) documented an 84 per cent increase in the occurrence of animal rabies in the United States in 1980 compared to the average for the preceding five years. Although this increase occurred predominantly among the wild, or sylvanic, animal population, the United States has seen a gradual increase in the occurrence of rabies among domestic animals since 1978.[2] A parallel increase in rabies positive wild and domestic animals has also occurred in Kansas *(Figure 1)*. This ominous increase in the isolation of rabies positive animals, especially in the domestic population, may present a great rabies risk to humans.

Although there were no reported cases of human rabies in the United States during 1980, two documented cases occurred in 1981.[3, 4] The health care community should not be lulled into a false sense of security because only two cases of rabies occurred during this two year period. In 1979, five cases of human rabies were reported to the CDC — more than were reported in any year since 1959. To complicate the picture, it is possible that patients thought to have died from atypical encephalitis may in fact have succumbed to rabies. Without a high degree of clinical suspicion, the diagnosis of rabies may be delayed or even missed. A good example of this problem is the most recent reported case of rabies identified in Kansas (1968).[5] A 13-year-old male presented initially with non-specific symptoms

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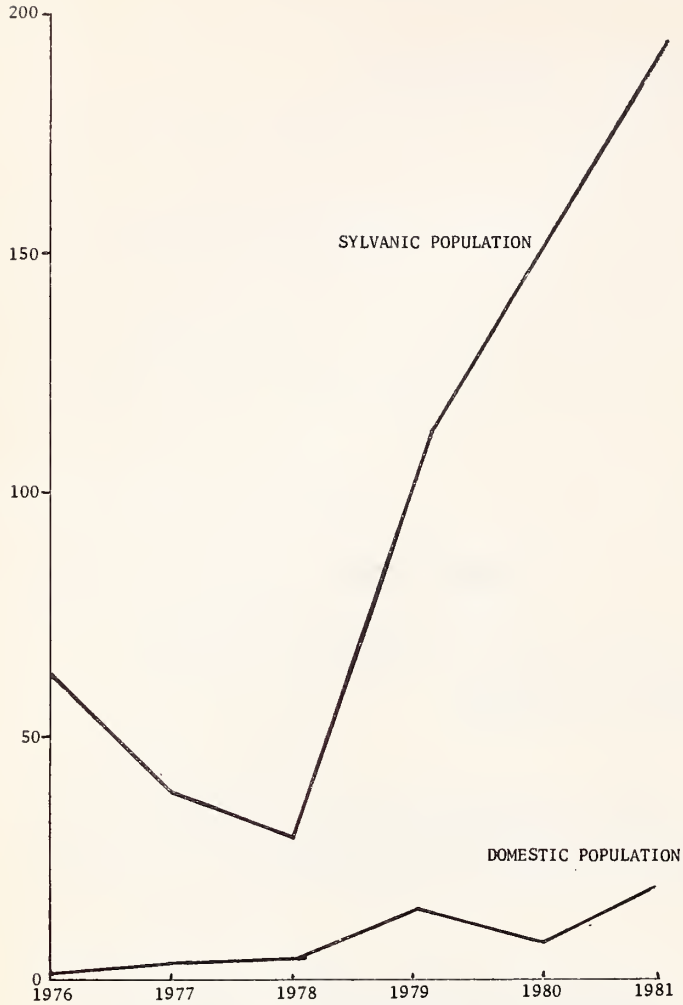


Figure 1. Rabies isolations, State of Kansas, 1976-1981.

that developed into a viral encephalitic representation of unknown etiology. Because the patient had no history of exposure to rabies, the initial differential diagnosis included poliomyelitis and other enterovirus diseases, Western equine encephalitis, California encephalitis, St. Louis encephalitis, mumps, and Rocky Mountain spotted fever. However, a rising rabies neutralizing antibody titer supported the diagnosis of rabies, and a positive rabies fluorescent antibody in post mortem tissue analysis confirmed the diagnosis.

The primary care physician is frequently called upon to evaluate a patient claiming to have been bitten, scratched, or licked by a potentially rabid animal. The physician needs to evaluate a series of variables to ascertain the risk of the patient contracting rabies. From the evaluation of these variables, a plan for the appropriate therapeutic intervention, if necessary, can be formulated.

The purpose of this article is three-fold: (1) To present guidelines for establishing patient risk of contracting rabies from an animal bite and evaluating criteria for initiating treatment; (2) To present current antirabies treatment protocols; and (3) To indicate where medical support and rabies information can be obtained in Kansas.

It is known that rabies virus in humans has a variable incubation period that ranges from ten days to one year. Most commonly, the incubation period for the virus is three to ten weeks. The reason for this variability of incubation is thought to be due to numerous factors; two of the most important include the location and the severity of the bite. Since the virus must travel up the peripheral nerves before it multiplies in the central gray matter, the incubation period may vary with the size of the viral innoculum (severity of the bite) as well as the distance that the virus has to travel to reach the central nervous system (location of the bite).

The prodromal signs and symptoms of the disease in humans are usually non-specific and include fever, malaise, insatiability, headache, and sore throat. Abnormal neurological manifestations referred to the bite site — such as pain, tingling, burning, cold sensation, pruritus, and paresthesia — may be of some diagnostic value. The patient soon enters the excitation phase in which anxiety and apprehension predominate. Clinical signs of hydrophobia may be seen at this time. When the patient tries to eat or drink a spasmodic discharge of respiratory and swallowing muscles ensues. The patient may succumb during one of these episodes or may progress to the paralytic phase in which flaccid paralysis slowly merges into coma and death.

When a human victim of animal bite seeks medical care, the physician must decide either to initiate or defer antirabies prophylactic treatment by carefully evaluating the following criteria:[6, 7]

- Prevalence of rabies in the geographic area
- Species of biting animal
- Severity and location of the bite
- Provoked *vs* nonprovoked bite
- Animal health status after veterinarian examination of the animal
- Vaccination status of the animal

See *Table I* for treatment flow sheet.

Prevalence of rabies in the geographic area. Skunks are the most commonly isolated rabid animals in Kansas and comprise 86 per cent of all rabies positive animals isolated during the last six years *(Figure 2).* Dogs, cats, cattle, foxes, and raccoons comprise less than 10 per cent of all rabies positive

TABLE I
TREATMENT FLOW SHEET

Animal Not Secured		*Animal Secured*		
High Risk of Rabies: in species of biting animal (raccoon, bat, skunk, fox, etc.).	Low Risk of Rabies: in species of biting animal (dog, cat, rabbit, rat, mouse, chipmunk, bird, etc.).	Consult with veterinarian and review the behavior of animal, risk of rabies in sylvanic and domestic animal population in regional area, if bite is provoked *vs* nonprovoked, and vaccination status.		
1. Treat with Human Diploid Cell Vaccine and Rabies Immune Globulin immediately.	Consult with veterinarian.	1. Abnormal behavior suggestive of rabies or 2. High risk of rabies in animal (bat, raccoon, skunk or fox) or 3. Nonprovoked bite.	1. No abnormal behavior. 2. Low risk of rabies in animal. 3. Provoked bite.	
2. Continue search for animal.	If veterinarian does not present conflicting epidemiological data and no abnormal animal behavior is noted: *Do not treat.*		*Sylvanic Animal*	*Domestic Animal*
3. Determine anti-rabies antibody level 2-3 weeks after last Human Diploid Cell Vaccine dose if suspect patient is immunocompromised.	If abnormal animal behavior documented, establish independent risk variables. Treatment may be indicated.	A. Kill animal and perform antirabies fluorescent rabies antibody test.	A. Kill animal and do FRA test.	A. Quarantine 10 days.
If animal is located and antirabies fluorescent antibody test is negative, discontinue antirabies prophylaxis protocol.		B. Commence treatment with RIG and HDCV.	B. Do not initiate treatment unless FRA test positive.	B. Do not treat. If abnormal behavior appears during quarantine, kill animal, get antirabies fluorescent antibody test and start treatment. If antirabies fluorescent antibody test positive, complete treatment.
If antirabies fluorescent antibody test is positive, complete Human Diploid Cell Vaccine protocol.		C. If antirabies fluorescent antibody test positive, complete treatment protocol, determine antirabies antibody titer 2-3 weeks after last Human Diploid Cell Vaccine if suspect patient is immunocompromised.		
		D. If antirabies fluorescent antibody test is negative, discontinue treatment protocol.		

animals in Kansas during this same period of time. As previously noted, there has been an increase in the absolute number of rabies positive domestic animals seen in Kansas. In the future, this increasing rate of domestic animal positivity may present the greatest risk of rabies to residents of this state.

Species of animal. Certain sylvanic animals constitute a reservoir of rabies virus. These animals include bats, skunks, foxes, and racoons. A bite exposure by one of these animals presents a significant rabies risk to the patient and thus appropriate antirabies prophylactic regimen should be initiated. Any wild animal involved in a bite incident should be immediately killed and sent to the state veterinary diagnostic laboratory for a rabies examination. If the subsequent fluorescent rabies antibody test is negative, the antirabies treatment can be discontinued. However, if the animal cannot be tested or if the fluorescent antibody test is positive the entire course of treatment should be carried out.

Vaccinated dogs and cats present a low risk of transmitting rabies to humans, but even in the face of a rabies vaccination history, each and every bite must be evaluated. All non-vaccinated domestic

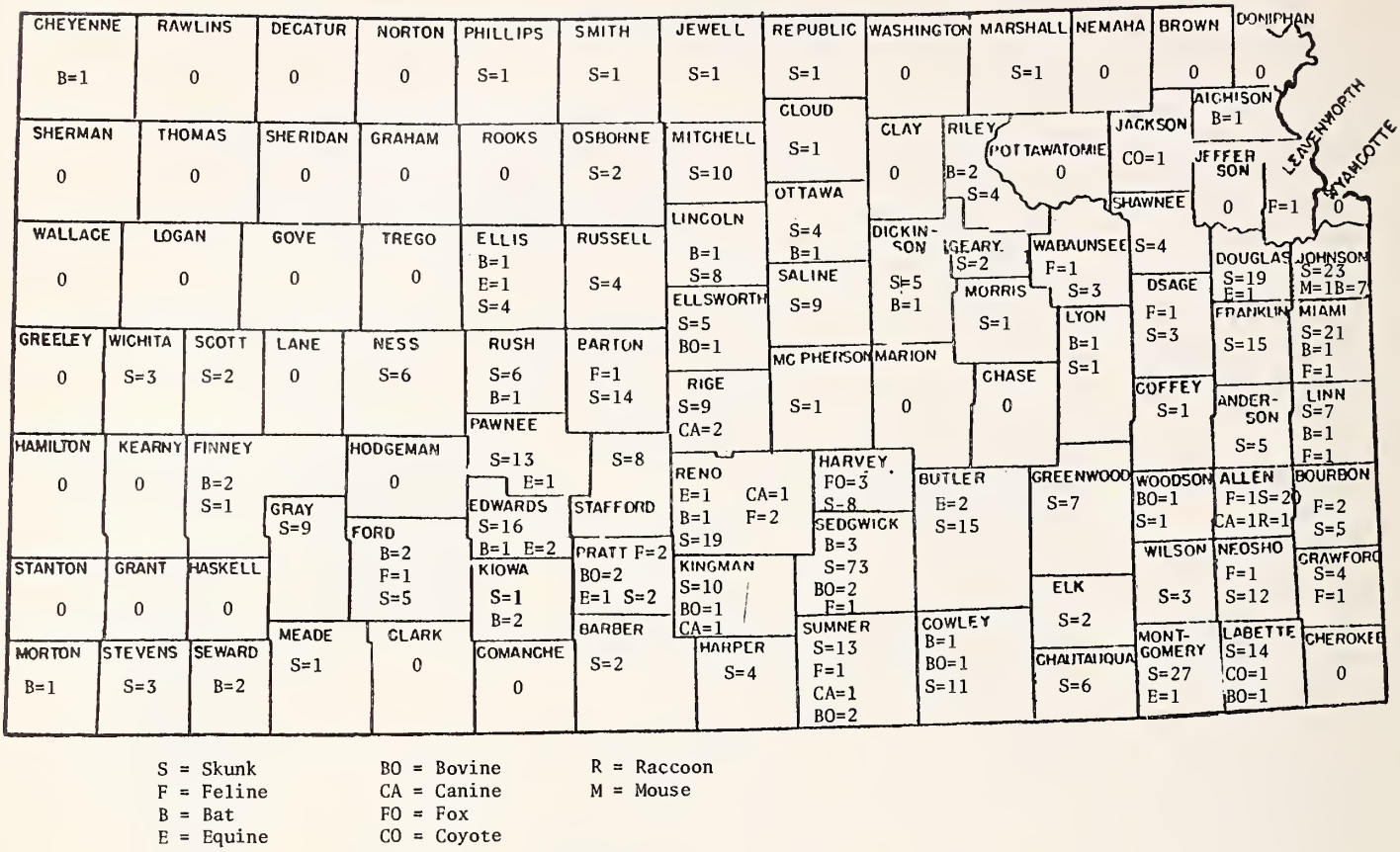


Figure 2. Rabies isolation by counties, State of Kansas, 1976-1981.

animals should be placed on a ten day quarantine under the supervision of a veterinarian. Vaccinated domestic animals, however, should be initially evaluated by a veterinarian and then may be placed on a ten day home quarantine with appropriate veterinarian followup. If any signs of rabies develop in the animal, it should be killed and a fluorescent rabies antibody test performed *(Table II)*. If abnormal animal behavior consistent with rabies is seen, the physician should be notified so the patient can be placed on the appropriate antirabies treatment protocol.

Severity and location of the bite. The most common documented method of contraction of rabies is by innoculation of saliva during a biting incident. Although rabies virus may penetrate a mucous membrane, the organism does not seem able to penetrate intact skin. In this latter instance, care must be exercised when verifying supposedly intact skin since minute scratches and abrasions will provide a portal of entry for the virus. Discretion may be the better part of valor when evaluating "intact skin." In these instances, antirabies prophylaxis may be indicated even though a break in the skin cannot be demonstrated.

Provoked vs nonprovoked bite. A provoked bite is defined as the expected pattern of behavior when an

TABLE II

SUBMISSION OF ANIMAL HEADS FOR FLUORESCENT RABIES ANTIBODY TESTING

Notification: Before shipping notify the Veterinary Diagnostic Laboratory, Veterinary Medical Science Building, Room 103, School of Veterinary Medicine, Kansas State University, (913) 532-5650 or 532-5690

What to Ship: Ship head of large animal or entire carcass of small animal.

How to Ship: Ship speciman using *wet ice*. DO NOT USE DRY ICE. DO NOT FREEZE. Wrap head or carcass in absorbant material and then place in two water tight plastic bags.

Note: USE CAUTION WHEN HANDLING. Always use gloves and masks when preparing the head or carcass.

animal is threatened, teased, or startled. In addition, a bite response may be expected when an individual threatens injury to a pet owner or a family member. However, an unprovoked bite — especially in a wild animal — is suggestive of the presence of rabies. Caution should be used in labeling a bite unprovoked when evaluating a wild animal attack or taking a history from a small child. Careful elicitation of an adequate biting history will often show the so-called "unprovoked bite" to be a provoked bite.

Veterinarian examination. A veterinarian should examine the biting animal immediately following the biting incident as well as during the ten day quarantine. It should be emphasized that the description of the animal's behavior by the patient may not be reliable. This is especially true when evaluating wild animal bites. It is well known that behavior patterns of wild animals differ considerably from those of domestic animals. Often, a rabid wild animal will allow a human to approach, pet, or capture it. Any abnormal behavior suggestive of rabies should be reported expeditiously to the primary care physician by the veterinarian. Subsequently, the animal should be killed for the fluorescent rabies antibody testing and results communicated to the physician so that therapy may be continued or terminated.

Vaccination status of the animal. Current vaccination protocols in the United States for domestic animals — especially dogs and cats — are highly effective in preventing the development of rabies. However, care must be used when evaluating the vaccination status of the animal. Veterinarian verification of up-to-date vaccination must be documented in all cases. Even if domestic animals have a positive vaccination history, they should be placed on a ten day quarantine. All wild animals are considered non-vaccinated since no rabies vaccine has been approved for use in the wildlife population.

Treatment Procedures

With the recent introduction of the Human Diploid Cell Vaccine (HDCV), many of the serious side effects of the Duck Embryo Vaccine (DEV) have been eliminated. Prior to the introduction of HDCV, the decision to treat or not to treat borderline cases was complicated by the adverse reactions often ascribed to the DEV. This problem of major side effects has now been resolved with the new treatment protocols available.

Although the cornerstone of antirabies treatment is the use of Rabies Immune Globulin (RIG) for immediate protection and Human Diploid Cell Vaccine (HDCV) for sustained antirabies antibody protection, a simple but important procedure should be remembered. The basic concept of wound cleansing is apropos in all cases. The mere act of prompt wound cleansing may substantially reduce the risk of rabies to the patient.

Bite Case Treatment Procedures

General: All bite wounds and scratches should be thoroughly cleansed with copious amounts of soap and water. If performed soon after the biting incident, studies have indicated that simple wound flushing significantly reduces the risk of contracting rabies by decreasing the amount of the viral innoculum. In addition, prophylactic antibiotics may be indicated in some dog bites, especially when the bite involves the hands or if a large area is severely traumatized.[8] Dog bite wound infections have been ascribed to numerous bacterial agents including Pseudomonas, Staphylococcus, Pasteurella, and Streptococcus. Antibiotic therapy should be directed against these organisms. In addition, a history of the patient's tetanus status should be ascertained and, if the patient is at risk for development of tetanus, appropriate intervention should be initiated.

Antirabies prophylaxis: The keystone of antirabies prophylaxis involves the use of both passively administered antibodies (Rabies Immune Globulin) and active immunization (Human Diploid Cell Vaccine). Unless the patient has been previously treated and has a documented adequate rabies antibody titer, Human Diploid Cell Vaccine should be given in a 1 ml dose on the day of the incident with subsequent intramuscular injections on days 3, 7, 14, and 28. In addition, Rabies Immune Globulin (RIG) can be given up to the eighth day after the first dose of HDCV is given. RIG is given only once, and the dosage is 20 international units per kg. One half of this dose (ten international units per kg) is injected into the wound site and the second half (ten international units per kg) is given intramuscularly. A serum specimen to determine the antirabies antibody titer should be drawn two to three weeks after the last dose of HDCV if the patient is immunosuppressed *(Table III).*

Local adverse reactions to HDCV occur approximately 25 per cent of the time and include pain, erythema, swelling, and itching. Systemic reactions such as headache and nausea occur less frequently. Serious side reactions are rare compared to those seen when using Duck Embryo Vaccine. It should be noted that immunosuppressive drugs may interfere with the patient's production of antirabies antibodies. Pregnancy is not considered a contraindication for post exposure treatment with either Human

TABLE III
TREATMENT PROTOCOLS

Prior Treatment (Previously documented adequate antirabies antibody titer)	*No Prior Treatment or Prior Treatment With Inadequate antirabies Antibody Titer*
Two one ml doses Human Diploid Cell Vaccine IM on days 0 and 3.	Five one ml dose of Human Diploid Cell Vaccine on days 0, 3, 7, 14, 28
Do not give Rabies Immune Globulin.	Rabies Immune Globulin, give once (up to day #8): 10 IU/Kg/IM 10 IU/Kg/infiltrate at site of wound.

Diploid Cell Vaccine or Rabies Immune Globulin.

Most counties in Kansas have established a County Health Department. Additional questions concerning the prevalence of rabies, isolation of suspected animals, and treatment can be directed to these local offices *(Table IV)*. In addition, all confirmed cases of human rabies must be reported to the County and State Health Departments.

TABLE IV
COUNTY HEALTH DEPARTMENT OFFICIALS
Additional information concerning rabies can be obtained from the County Health Department

County	*Telephone*	*County*	*Telephone*
Allen	316-365-5514	Linn	316-365-5514
Anderson	316-365-5514	Logan	913-672-4502
Atchison	913-742-7192	Lyon	316-342-4864
Barber	316-886-3294	Marion	316-382-2550
Barton	316-793-7879	Marshall	913-562-3485
Bourbon	316-365-5514	McPherson	316-241-1753
Brown	913-742-7192	Meade	No Hlth Dept
Butler	316-321-3400	Miami	913-294-2431
Chase	316-273-6377	Mitchell	913-738-5175
Chautauqua	316-725-3300	Montgomery	316-251-4210
Cherokee	316-429-3087	Morris	316-767-5175
Cheyenne	913-332-2381	Morton	316-697-2612
Clark	316-635-2624	Nemaha	913-284-2288
Clay	913-632-3646	Neosho	316-244-5640
Cloud	913-243-3588	Ness	913-798-3388
Coffey	316-364-8631	Norton	913-877-2810
Commanche	316-582-2431	Osage	913-828-3281
Cowley	316-221-1430	Osborne	913-346-2412
Crawford	316-231-5411	Ottawa	913-392-2822
Decatur	913-475-3090	Pawnee	316-285-6963
Dickinson	913-263-4179	Phillips	913-543-2179
Doniphan	913-985-3886	Pottawatomie	913-457-3719
Douglas	913-843-0721	Pratt	316-672-7436
Edwards	316-659-2583	Rawlins	913-626-3968
Elk	316-374-2277	Reno	316-663-6721
Ellis	913-625-2013	Republic	913-527-5385
Ellsworth	913-472-4234	Rice	316-257-2171
Finney	316-276-3281	Riley	913-776-4779
Ford	316-225-4991	Rooks	913-425-7352
Franklin	913-242-1873	Rush	913-222-3427
Geary	913-762-5788	Russell	913-483-2641
Gove	913-938-2335	Saline	913-827-9376
Graham	913-674-3326	Scott	316-872-5774
Grant	316-356-1545	Sedgwick	316-268-8241
Gray	316-855-2424	Seward	316-624-3742
Greeley	316-376-4200	Shawnee	913-233-8961
Greenwood	316-321-3400	Sheridan	913-675-2101
Hamilton	316-384-7875	Sherman	913-899-3625
Harper	316-842-5264		X201
Harvey	316-284-2082	Smith	913-282-6661
Haskell	No Hlth Dept	Stafford	316-549-3504
Hodgeman	316-357-8736	Stanton	316-492-6443
Jackson	913-742-7192	Stevens	No Hlth Dept
Jefferson	913-863-2447	Sumner	316-326-2774
Jewell	913-378-3976	Thomas	913-462-3335
Johnson	913-384-1100		X54
	X283	Trego	913-743-6348
Kearney	316-355-6342	Wabaunsee	913-765-2280
Kingman	316-532-2221	Wallace	913-852-4272
Kiowa	316-723-2542	Washington	913-325-2600
Labette	316-421-4350	Wichita	316-375-2289
Lane	316-397-2333	Wilson	316-378-2324
Leavenworth	913-682-0245	Woodson	316-365-5514
Lincoln	913-524-4709	Wyandotte	913-321-4803

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Knee Injuries

Damage From Running and Related Sports

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The average physician will encounter more running-related injuries than any other type of sports injury. The knee is the most common site of injury for both men and women, and knee damage is probably the most serious disability that can result from running. Most injuries result from improper scheduling, equipment, and running surface.

Millions of Americans have taken up running for its physical and psychological benefits, and for plain enjoyment. It is estimated that about 11 million Americans are participating in running (or jogging).[1] The average physician will encounter more patients with running-related injuries than any other type of sports injury. Many of these injuries are not serious — minor strains and sprains — but some are major and permanent. Knee damage probably represents the most serious disability as a result of running.

In a typical runner's clinic, 30 per cent of the running injuries involve the knee joint.[2] The knee is the most common site of injury for both men and women. The risk of injury increases with increasing weekly mileage.[1]

Of human joints, the knee joint has the largest area of articular cartilage and synovial membrane. It is the most complicated in terms of mechanics;[3] flexion is successively a combination of rotation, arc, and gliding movements far removed from the concept of a simple hinge. It occupies an exposed position at the junction of the longest and the strongest levers in the body. Of the weight-bearing joints, it is the most vulnerable to trauma, incidental or repetitive in the form of wear-and-tear. There are few disease processes affecting joints to which it is not the most susceptible joint.

Mechanics of Injuries

Training errors are the most frequent cause of running injuries.[4] Excessive mileage, intensive work-outs with interval training, and a rapid increase in mileage overwhelms the body's ability to adapt to new levels of stress. Insufficient warm-up can also over-stress joints. A rapid change in training routine such as alternately running fast and jogging on a track or running on hills and hard surfaces can also cause injury.

Shin splints, chondromalacia, muscle soreness, hamstring strain, and low-back pain are commonly seen because of poor training techniques and improper shoes. Stress fractures are also occasionally seen in the runner because of continuous repetitive trauma, particularly if the runner is not sufficiently trained and is using improper shoes for running on an irregular surface. The overuse syndrome occurs when the runner progresses from one level of training to another, increasing mileage or speed. The runner simply overuses his body, not allowing it to adapt to a higher level of stress.

Proper shoes are essential to avoid running injuries. Wearing poorly-constructed running shoes or inappropriate shoes, such as sneakers, can contribute to injury as they do not provide the necessary cushioning or stability needed to protect the joint over the foot and leg from the repetitive shock of running. Repair of running shoes is important as well, because excessive wear on the outer heel creates an imbalance as the heel strikes that may cause lateral knee pain.

The running surface is important. A soft, level surface, such as a dirt path is ideal. Most people run on whatever surface is most convenient to them. Running on concrete sidewalks and up-and-down curbs exaggerates the shock transmitted to the legs, feet, knees, and back. An asphalt road, on the other hand, may provide more cushioning. As grassy surfaces are irregular and sand unstable, such surfaces are not recommended for running or jogging.

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Evaluation of Knee Injuries

The secret of success in knee evaluation is thoroughness. This applies particularly to the history because, in many cases of internal derangement, physical examination reveals no sign upon which diagnosis can be based.

It is well-known that *pain* — the most reliable and yet unaccessible of symptoms — is the commonest and most important single reason for seeking advice. The patient's opinion of the site of the pain is of diagnostic importance in determining the derangement; for example, lateral knee pain, pain in the infrapatellar fat pad, patellofemoral joint pain, all give clues to the primary anatomical area at fault.

A sudden *swelling* of the entire joint within, say, one-half hour of injury is a hemarthrosis. Insidious swelling of the entire joint is likely to have a more serious pathological background and often provides considerable problems of diagnosis.

Stiffness may be defined as resistance to mobility. There are normal physiological variations to this sensation; it is greatest at the beginning and at the end of the day. It is also a symptom, but not the major complaint, in all internal derangements of degenerative origin.

Insecurity (giving way) is a common symptom that results from the intraposition of some small object (as opposed to a large one, as seen in locking) in the form of a fragment of meniscus, articular cartilage, or loose body between opposing articular surfaces under circumstances of stress. In the patellofemoral joint, particularly in the presence of a flexion deformity, a tag of articular cartilage from chondromalacia is the likely obstructive object. It is also possible that the symptom is related to no more than the existence of a minor flexion deformity and the related quadriceps wasting.

Locking is a symptom associated with the young male athlete and is defined as a sudden and complete painful block to extension sustained by the mechanism of undue rotation and flexion, usually in some contact game like football or soccer. The torn meniscus is caught between the femoral condyle and the upper surface of the tibial plateau.

Examination of the patient with a knee injury requires complete relaxation of the patient. A patient required to lie on his/her back while the knee is examined will almost invariably raise the head to see what is going on. It should be explained that relaxation is important and the head should remain on the pillow. The human eye is the most sensitive instrument of clinical examination of the knee joint. It can detect what cannot be palpated, measured, or otherwise recorded. The first action is to compare the development of the quadriceps on both sides.[5] The muscle should be observed first in relaxation and then in contraction, as the patient is asked to brace his knees against the couch. If a difference is noted, it is pertinent, in a youth, to inquire if he is naturally right or left-footed. Differential wasting involving vastus medialis alone could be the clue to the diagnosis of recurrent subluxation of the patella. If there is no wasting of the quadriceps, there is not likely to be very much wrong with the knee.

The examiner should look for surgical scars, erythema, and effusion. Knee effusion in a runner who has just run seven miles or more may present a mild reactive synovitis that is not necessarily pathological.

Many of the disease processes of the patellofemoral joint are closely associated with the relationship of the patella to the femoral condyles. The approach to diagnosis of disturbance here is observation first with the patient erect and then supine, to determine the position of the patella. The presence of patella alta, or lateral position of the patella, or any other abnormality should be recorded.

The Q angle is measured by drawing lines from the anterior superior iliac spine and from the tibial tubercle through the midpoint of the patella. A Q angle greater than 20 degrees is considered abnormal.[6] Other findings associated with patellar pain are medially directed or laterally directed patella, lateral insertion of the patellar tendon into the tibial tubercle, and patella alta.

A synovial effusion outlines the horseshoe-shape of the suprapatellar pouch and is detected by the experienced person at a glance. With minor effusion, patella tap is not normally performed, as cross-fluctuation is the best way to palpate for synovial effusion. Synovial thickening can be distinguished from effusion by finger palpation of the margins of the suprapatellar pouch. Local tenderness is an important feature of lesions of the infrapatellar fat pad, and of traction epiphysitis of the patellar tendon in youth, either at the tibia (Osgood-Schlatter's disease) or at the patella (Sinding-Larsen-Johansson's disease). The examiner should also test for increased heat in the joint by comparing the two sides. The examiner should measure the degree of flexion and extension, and test for any anteroposterior, mediolateral, or rotary instability.

Plain x-rays are essential in the evaluation of many running injuries. The AP, lateral, tangential ("sunrise"), and tunnel views are needed in diagnosing knee problems, especially pre-existing conditions, malalignment, or patellar-tracking prob-

lems. Arthrography is used to diagnose intra-articular damage such as a tear in the meniscus. However, as rotational injuries are rare in runners, arthrography should be used only if one suspects such an injury has occurred.

Chondromalacia Patellae

By far the most common knee problem in runners is chondromalacia caused by a tracking abnormality of the patella and frequently referred to as runner's knee.

Chondromalacia is a degenerative process of the articular cartilage of the patella and opposing femoral condyles characterized by fibrillation, fissuring, and eventual erosion exposing bone. In a later form it will be recognized as osteoarthritis of the patellofemoral joint.

It is difficult to explain chondromalacia in the teen-ager or young adult in the absence of specific trauma, other than proposing a congenital inherent defect in the articular cartilage. Certain anatomic and biomechanical factors affect the normal tracking of the patella. Tightness of the vastus lateralis, iliotibial tract,[7] and lateral retinaculum, coupled with weakness of the vastus medialis, move the patella laterally during flexion[8] and cause pain on contraction of the quadriceps. The malalignment syndrome is also associated with lateral displacement of the patella. The increased Q angle produces a bowstring effect on the quadriceps, pulling the patella laterally as the quadriceps contracts, at heel strike, and as the tibia rotates internally on the pronated foot in midstance. Patella alta also predisposes the patella to lateral stresses and instability. The typical patient with chondromalacia is a novice or sports runner who is increasing his/her mileage. Sometimes this condition results from overuse, as in the classical example of the professional long-distance cyclist. On the other hand, disuse in extreme form causes greater articular cartilage degeneration than overuse. An extreme example of disease entails immobilization in a plaster cast or caliper. Cutting a window in the cast over the front of the patella, to relieve pressure of the plaster cast on the patella, would prevent the development of chondromalacia of the patella.[9]

Chondromalacia patellae can be classified into four grades (Outerbridge, 1961).[10]

Grade I: Localized softening, swelling or fibrillation of the articular cartilage.
Grade II: Fragmentation and fissuring in an area 1.3 cm or less in diameter.
Grade III: Fragmentation and fissuring in an area of more than 1.3 cm in diameter.
Grade IV: Erosion of articular cartilage down to subchondral bone.

Clinical Features

The presenting feature is pain or ache experienced in or under the patella. It is produced by movements that oppose patella and femur under compression, as in rising from a chair or ascending stairs, or hill-running. Patients complain that the joint is stiff or they feel some obstruction that tends to catch, cause momentary locking, or otherwise interfere with smooth movement. Stiffness after sitting or "giving out" of the knee may also bring the runner to the physician. In chondromalacia patellae, there is no synovial swelling nor effusion. If synovial effusion is a feature, it is probably not chondromalacia because effusion is the result of irritation of synovial membranes, not irritation of articular cartilage. In any event, the symptoms of chondromalacia tend to disappear when an effusion occurs, as the patella is floated off the femoral condyles. The patient may exhibit the malalignment syndrome of the patella, a poorly-developed vastus medialis, "squinting" patellae,[4] joint crepitus, or mild effusion insufficient to float the patella. If the examiner attempts to move the patella laterally, the patient contracts the quadriceps, a reaction called a positive anxiety or apprehension sign. Extending the knee from 30 degrees of flexion to full extension against resistance reproduces the pain, as does compression of the patella against the femoral condyles.[11] There may be synovial tenderness at the margin of the patella, particularly on the lateral side.

Standard x-rays may appear normal, but skyline view may reveal an increased femoral sulcus angle, with inadequately-developed lateral femoral condyle with lateral displacement or lateral tilt of the patella, giving a "jockey cap" appearance of the patella. On lateral roentgenogram, the relative length of the patella and the patellar tendon are measured to diagnose patella alta. A ratio 1:1 is normal, a difference of 1.0 cm is considered significant.[6] When the condition is advanced, loss of joint space, sclerosis, or subchondral bone and marginal exostoses may show up on x-ray and indicate the transition to osteoarthrosis. Arthroscopy, while not usually necessary, will show the fibrillated patellar cartilage and can be applied to confirm or refute a provisional diagnosis.

In the differential diagnosis, one may consider rheumatoid arthritis, osteochondritis dissecans, pigmented villonodular synovitis, and synovitis of the knee. Hip or pelvic abnormalities may produce re-

ferred pain via the obturator nerve as the anterior branch supplies the hip joint and the posterior branch terminates in the knee. This may be misinterpreted by the patient or physician as knee pain.[3]

Treatment

Reassurance should be given to the patient, with an explanation of the origin and simple nature of the pathology, to reduce or eliminate the anxiety aspect of the symptom complex. For the runner seen soon after the first appearance of symptoms, the treatment includes rest, application of ice to the knee until the skin turns red (5-7 minutes), and aspirin. Other anti-inflammatory medications will help alleviate symptoms. After 24-36 hours, ice treatment is discontinued, and moist heat is applied to the knee for 15-20 minutes several times for as long as needed.

Rest from running is essential, although the runner may say s/he can alleviate the pain by running with the feet turned-in or pigeon-toed, which decreases the Q angle. The runner can do this only until s/he becomes fatigued. The patient should also avoid putting the patellofemoral joint under compressive loading such as kneeling, excessive stair-climbing, and prolonged sitting or squatting. Immobilization is contraindicated in any form and, in particular, the use of a plaster cast is irrational and undesirable. If chondromalacia indicates the breakdown of cartilage nutrition, immobilization will aggravate the situation.[9] The application of a plaster cast will render the patient symptomless, but mobilization of the joint results in a return of pain — possibly in more intense degree from related reduction in muscle power. When the pain is gone, the patient begins progressive resistive exercises with the knee in extension to strengthen the quadriceps, especially the vastus medialis. The following exercises are recommended:[12]

1. Quadriceps setting: ten-second contractions for five minutes each hour while awake.
2. Straight-leg raising: fifteen non-stop repetitions with a maximum possible resistance each hour while awake.
3. Isometric or isokinetic knee flexion: fifteen repetitions.

Resistance exercises will be avoided within the range of flexion in which pain or interference with motion is experienced. Progressively-loaded straight-leg raising is advised; the weight is gradually increased to eight to ten pounds during a period of several weeks. When the patient can lift without pain 50 per cent of the weight he can lift with the uninvolved leg, he can begin lifting the weight while flexing the knee from 10-30 degrees. When he is asymptomatic during normal activities, the patient can begin flexing the knee from 10 to 90 degrees.

The runner may begin a graduated program of running when he can run without pain. Immediately after a run he should apply ice to the knee. Several hours later, moist heat should be applied. Continued weight-training and avoiding all hill-running are essential to avoid recurrence.

Knee wraps, infrapatellar straps (a simple brace that encircles the knee joint below the patella, devised to lift the patella off the condyles),[13] and derotation braces (Lenox-Hill brace) have been used to relieve symptoms. These may be superceded by a well-tolerated orthotic device (molded insole orthosis) for the shoe. Intra-articular injection of steroids may alleviate the early symptoms. However, runners rarely accept this measure, preferring correction of the biomechanical problem with exercise and orthotic devices. Surgery is rarely performed for this condition in runners. In most recurrent problem cases, proximal realignment with lateral retinacular release, medial capsular reefing, and vastus medialis advancement may be considered. Shaving of the abnormal articular cartilage until normal tissue is reached may be combined with longitudinal division of the lateral capsule, with the purpose of relaxation of tension such as may be affected by the degree of realignment which occurs. In patella alta it may be necessary to transplant the tibial tubercle either to the medial side or to the medial side at a lower level.[14] In the adolescent with recurrent chondromalacia, with or without subluxation, correction of the biomechanical problem to allow running should be done if the child can run painlessly. It may, however, be wiser to direct the child to another sport.

Jumper's Knee

Osteochondritis of the inferior pole of the patella is a condition in young adults with clinical manifestations of Sinding-Larsen-Johansson disease, but without related radiological changes. The condition is commonly called "jumper's knee";[3] it may also be referred to by a variety of other names such as high-jumper's knee, basketballer's knee, volleyballer's knee, cross-country knee, place-kicker's knee, high-diver's knee, triple-jumper's knee, thus relating it to a variety of sports in which the extensor apparatus malfunction may be dubbed "tennis elbow of the knee." The pathology is unknown; it is assumed, however, that there arises the possibility of a deficient blood supply at the osseoustendinous

junction. An elongated inferior pole may be subject to a stress fracture.

Clinical Features

The salient feature is pain of insiduous origin located at the inferior pole of the patella. There is local swelling and tenderness. Symptoms are disabling and interfere with participation in the related sport. Occasionally a patient complains of pain over the patellar tendon on moving and of stiffness after sitting for any length of time, indicating patellar tendinitis. When gentle pressure is exerted over the tendons, a creaking sensation is expected. It is very similar to tenosynovitis crepitans about the wrist.

The treatment is unsatisfactory to both patient and physician. Such measures as heat, cold, exercise, and support may give temporary relief, but have no lasting effect on pain induced by the related sport. The temptation to inject hydrocortisone should be *strongly resisted,* as it is liable to contribute to a catastrophic total rupture of the tendon. The condition is self-limiting and, if untreated, symptoms may persist for as long as four years. Multiple drill holes in the inferior patellar pole through a short lateral parapatellar incision, in the hope of improving the local blood supply, may be tried. As a last resort, resection of the inferior pole with resuture of the tendon, as for a fracture, may be performed.

Occasionally in a patient with pain at the inferior pole of the patella, an ununited fracture is revealed on x-ray. It may be difficult to determine whether the ununited fragment is the result of an anomaly of ossification, Sinding-Larsen-Johansson disease, or a stress fracture superimposed on an elongated inferior pole as the result of ischemia. If symptoms persist, excision of the ununited fragment through a short, lateral parapatellar incision can be done.

Iliotibial Band Friction Syndrome

The iliotibial tract (band) is a thick strip of fascia latae that presses down the lateral aspect of the thigh and inserts into Gerdy's tubercle on the lateral tibial condyle. As the knee flexes and extends during running, the iliotibial tract repeatedly rubs over the lateral femoral condyle, which may cause an inflammatory response.[15] This produces lateral knee pain above the joint, but not as localized as the pain of popliteus tendonitis.

This syndrome usually occurs in novice runners with tibia vara and hyperpronated feet. The symptoms may also occur when a runner fails to repair a worn outer sole of a running shoe, and frequently may disappear when the shoe is repaired. Pain and tenderness at the insertion of the iliotibial tract into Gerdy's tubercle may also occur from overstress.

Therapy includes application of ice to the knee for up to 20 minutes, followed by exercises to stretch the iliotibial tract. Anti-inflammatory medications and injection of steroids into the area of maximum tenderness may be helpful. Proper shoes or shoe orthotic devices may control the forces generated through the lateral aspect of the knee at heel strike and with internal rotation of the foot. If the lateral sole of the shoe is worn, it must be repaired or the shoe replaced.

Popliteus Tendonitis

Popliteus tendonitis is directly related to hyperpronation and to running downhill. It is frequently seen in runners who run along the beach or other banked surface, as this increases the foot pronation on the higher part of the slope. Concomitant internal tibial rotation applies traction to the attachment of the popliteus tendon to the lateral femoral epicondyle. As the popliteus acts as a checkrein to prevent the femur from displacing forward on the tibial plateau during mid-stance, running downhill puts excessive stress on this structure. The pain develops acutely at the insertion of the popliteus tendon into the lateral femoral epicondyle.

Physical examination reveals tenderness over the tendinous portion of the popliteus, just anterior to the origin of the fibular collateral ligaments from the femur.[16] The patient is examined while supine; the affected knee is flexed at least 90°; the hip is flexed, abducted, and externally rotated; and the foot is placed on the knee of the other leg. Localized pain at the attachment of the popliteus tendon will differentiate this injury from other conditions producing lateral knee pain such as the iliotibial band friction syndrome or a pre-existing tear, cyst, or degeneration of the lateral meniscus.

The treatment for popliteus tendonitis is the same as that for iliotibial band friction syndrome, and includes correction of the biomechanical problem or a change in running surface and conservative measures to alleviate pain.

Summary

The most common knee problems and injuries as a result of running and similar sports are chondromalacia patellae, jumper's knee, iliotibial band friction syndrome, and popliteus tendonitis. The underlying biomechanical problems, the pathological anatomy, and presenting clinical symptoms and signs, and

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PCP Intoxication

Management of Associated Renal Failure

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Phencyclidine (PCP) intoxication may result in rhabdomyolysis, and acute renal failure may readily occur. Currently advocated therapy protocols may, under some circumstances, precipitate acute renal failure. The case described here illustrates common clinical and therapeutic features associated with the advent of renal failure.

Phencyclidine Hydrochloride (PCP, Angel Dust) is a commonly abused drug, and clinical intoxication is reported with increasing frequency. Abuse results in unusual psychosis, thought disorders, agitation, sympathetic dysfunction, respiratory depression and coma, and drug related death which usually occurs as a result of asphyxia, drowning, trauma, suicide or homicide.[1] Rhabdomyolysis and myoglobinuria have previously been reported in other cases of drug abuse related to heroin,[2] amphetamines[3] and in three cases of phencyclidine abuse.[4, 5] Barton *et al.*[6] recently called attention to the occurrence of acute renal failure in two cases of phencyclidine intoxication. We here report an additional case of myoglobinuric acute renal failure which occurred as a result of phencyclidine associated rhabdomyolysis.

Case Report

A 29-year-old black male was admitted to the Kansas City V.A. Medical Center emergency room in a combative state, unresponsive to verbal commands. Admission occurred within two hours after smoking PCP. In the preceding two months the patient had been admitted briefly three times for PCP detoxification, the last occasion one week prior to admission. During the initial examination the patient exhibited increasingly combative behavior with bizarre violent movements which necessitated physical restraints. In addition, there was profound diaphoresis with increased salivation. The patient then vomited profusely; aspiration could not be prevented. Blood pressure was 150/90 mmHg; pulse rate, 100/minute; respiratory rate, 28/minute; and temperature, 38.5 C. Physical examination was otherwise unremarkable. In particular there was no evidence of trauma, neck rigidity or papilledema; and pupillary light reflex, deep tendon reflexes, and muscle tone and activity were all normal.

Treatment in the emergency room included gastric lavage and standard supportive therapy used for coma patients. The patient also received 50 grams of dextrose intravenously as well as 0.4 mg naloxone hydrochloride (Narcan), and 10 mg diazepam intravenously, without any change in agitation or responsiveness.

The pertinent laboratory examinations revealed: sodium, 138 mEq/L; potassium, 3.1 mEq/L; chloride, 96 mEq/L; CO_2, 31 mEq/L; glucose, 137 mg/100 ml; creatinine, 1.2 mg/100 ml; BUN, 16 mg/100 ml; and calcium, 10.4 mg/100 ml. Urinalysis showed specific gravity 1.024, pH 6.5, 1 + glucose, and microscopic examination yielded normal results. Radiogram of the chest was normal. A lumbar puncture was performed with normal findings in the CSF fluid. Gastric aspirate revealed acetaminophen which later corresponded to a serum level of 10 mcg/100 ml. Mass spectroscopy confirmed the presence of large amounts of PCP in the urine.

Specific therapy included acetylcysteine which was given initially for the presumed acetaminophen intoxication after the drug had been identified in the gastric aspirate. This was discontinued when the nontoxic serum concentration became known. Urinary acidification with ammonium chloride and ascorbic acid was also started to improve urinary excretion of PCP as previously described.[7, 8]

During the initial ten hours of acidification treatment, arterial pH remained 7.34 with a urine pH of 6.0, but serum creatinine rose to 2.5 mg/100 ml. Arterial pH gradually decreased to 7.15 during the

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next 38 hours. Serum creatinine rose further to 4.9 mg/100 ml at the time that the urinary flow rate decreased to 70 ml/hour despite generous volume support. At that time the patient's urine also became cloudy and tested positive for myoglobin, and the serum CPK rose to 156,000. Acidification was discontinued. The patient also required mechanical ventilation for increasing hypoxia resulting from aspiration pneumonia.

During the next seven days the patient developed progressive fluid retention. BUN and creatinine rose to 105 and 15.5 mg/100 ml respectively. There was also clinical evidence of pericarditis. At this time the patient received four consecutive hemodialysis treatments. He recovered uneventfully from the acute renal failure and was discharged 24 days after admission with normal renal function characterized by a serum creatinine concentration of 1.1 mg/100 ml and a BUN of 21 mg/100 ml.

Discussion

Phencyclidine has been withheld from human use since 1965 and is now legitimately available only in the veterinary medicine practice for the tranquilization of large animals. However, among drug users it has found increasing popularity as a psychodelic or hallucinogenic agent.[9] It is frequently found in a mixture with other common street drugs; for example LSD, mescaline, or cannabis (marijuana). Most commonly it is available in the physical form of tablets but may also occur in a leaf mixture usually rolled as cigarettes (joint).[1, 10]

Clinical toxicity is characterized by a complex acute organic brain syndrome and increased sympathetic activity. However, renal failure may occur more frequently than is currently appreciated. One case of renal failure with PCP intoxication was previously attributed to vasculitis and consequently treated with steroids.[4] However, rhabdomyolysis was also present and it appears that rhabdomyolysis with myoglobinuria may readily result in renal failure. The potential occurrence of this complication was first discussed by Cogen *et al.* in 1978,[5] and then documented in two cases of non-oliguric renal failure reported by Barton *et al.* in 1980.[6] The present case again documents the occurrence of PCP related acute myoglobinuric renal failure in what appears to be a typical setting.

The common features in the cases of PCP related rhabdomyolysis described to date are: (1) central nervous system depression; (2) marked increase in muscular activity; (3) necessity for physical restraints during hospitalization; (4) mild hyperpyrexia; and (5) hypertension. In particular, the combination of excess physical activity with restraints may be the major factor in the occurrence of rhabdomyolysis. This is well documented in laboratory animals.[11] Denervation of animals resulted in decreased serum CPK activity. Likewise, peripheral nerve paralysis resulted in a decrease in muscle enzyme concentrations in one patient previously reported.[5]

Urinary acidification has previously been advocated as an adjunct in the treatment of PCP intoxication. It is used to enhance drug excretion because the drug has a pKa of eight to nine, and hence becomes more soluble in an acid environment.[7, 8] This treatment must now be reevaluated in light of the previous reports of rhabdomyolysis with and without renal failure and the current case report. Precipitation of myoglobin or hemoglobin pigments in renal tubules is increased by urinary acidification.[12] Medical management of pigment related renal failure rests exclusively on supportive measures, increased urinary flow rate, and urinary alkalinization. Treatment of acute severe intoxication with PCP should also be based on these general principles to maintain adequate urinary flow rate. Restraints for severe agitation should be avoided if at all possible to preclude rhabdomyolysis. When such agitation cannot be controlled adequately we recommend peripheral muscular blockade followed by forced diuresis and urinary alkalinization to prevent myoglobinuria. Urinary acidification may be of limited value in the treatment of PCP intoxication only in the absence of muscular agitation.

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Neuroblastoma in Children

A Retrospective Review and Update on Treatment

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Neuroblastoma is the fourth most common solid tumor in children. Three-fourths of cases occur before the age of 4 years. Surgery and radiation therapy are the most successful therapeutic modalities, but chemotherapy based on cell kinetics of the tumor has recently shown significant promise. Presented here are results of a ten-year retrospective study correlated with other studies and a discussion of newer insights into treatment.

NEUROBLASTOMAS are derived from neural crest tissue and are generally located at any anatomical site along the craniospinal axis. Neuroblastoma is a common childhood tumor, being the fourth most common solid tumor in children.[1] The incidence is approximately 9.6/million in white children and 7.0/million in black children.[1] Fifty per cent of cases of neuroblastoma occur before 2 years of age, and 75 per cent cases occur before 4 years of age.[2, 3] Patients most commonly present with an abdominal mass, but the tumor can present as masses in other areas causing local pressure symptoms. Patients also may present with symptoms related to catecholamine production such as fever, hypertension, diarrhea, skin flushing, and headaches. Certain factors postulated to have prognostic significance at the time of diagnosis include stage of disease, age of the patient, peripheral lymphocyte count, lymph node involvement by the tumor, and vanillylmandelic acid (VMA): homovanillic acid (HVA) ratio in the urine.[4, 5] Of these, the only two that have definite correlations with outcome are age of the patient and stage of disease at diagnosis.[4, 6]

Age at diagnosis is very important. Children less than 1 year of age have a distinctly better prognosis compared to older children, even in the presence of extensive metastatic disease (stage IV-S). Survivals of up to 80 per cent have been reported with stage IV-S disease. This is comparable with survivals in stage I and II. The reason for this phenomena is still speculative. The overall survival rate for neuroblastoma ranges from 13 to 31 per cent depending on the study.[2]

Accurate staging is critical for treatment planning of neuroblastoma. The stagings of Evans *et al.*[6] and of St. Jude's research group are shown in *Table 1*. The classification by Evans *et al.* is primarily clinical, whereas that of St. Jude's is a combination of surgical and pathological staging.

Diagnostic tests other than surgical exploration should include complete blood count, coagulation studies, bone marrow aspiration, and analysis of urine for catecholamine metabolites. The two major metabolites are homovanillic acid (HVA) and vanillylmandelic acid (VMA). These are present and elevated in as many as 80 per cent of patients with neuroblastoma.[5] Cystathionine may be excreted in patients with neuroblastoma. This metabolite is excreted also in cystathioninuria and liver diseases. Calcification is present in about half of the neuroblastomas. Roentgenograms of the chest, skeletal survey, bone scan, bone marrow examination, liver/spleen scan, and intravenous pyelogram are necessary for accurate staging. The intravenous pyelogram may show the classical "drooping lily" appearance *(Figure 1)*. This suggests that the lesion is extrarenal in origin, and can be distinguished from renal tumors which classically produce distortion of the pelvis and the calyces.

Neuroblastomas may present with varying grades of histologic maturation. Vesicular nuclei, cytoplasm and cytoplasmic processes, presence of mature ganglion cells, and rosette formation suggest an increase in maturity. Although attempts have been made to correlate these grades with clinical behavior of tumors, no definite correlation of histologic type with outcome has been observed.

The role of surgery in neuroblastoma is for biopsy, staging, sampling, and removal of primary tumors at diagnosis, or second look surgery for validation of clinical response, and possible "debulking" even though the role of "debulking" the

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TABLE I
STAGING OF NEUROBLASTOMA

Evans et al.		*St. Jude's Research Group*	
I	Tumor confined to organ or structure of organ	I	Tumor localized and completely resected
II	Tumor beyond organ or structure of organ, not crossing the midline (ipsilateral side lymph nodes may be involved)	II-A	Tumor localized and completely resected but microscopic spread through capsule
		II-B	Tumor localized, partial or no resection
III	Tumor beyond midline in continuity, bilateral regional lymph nodes may be involved	III-A	Disseminated tumor, without bone or bone marrow involvement
		III-B	Disseminated tumor, with one local bone lesion only
IV	Remote disease: bones, bone marrow, soft tissue, distant lymph nodes	III-C	Disseminated tumor, with bone marrow and/or generalized bone involvement
IV-S	Remote disease in liver, skin, or bone marrow, in otherwise stage I or II patients		

tumor is controversial.[7, 8] A complete excision of tumor at any age, in patients with non-metastatic disease, is associated with good prognosis. Patients with tumor involvement in lymph nodes generally do poorly.[7]



Figure 1. IVP showing "drooping lily" appearance suggestive of neuroblastoma.

The use of radiation therapy is not clearly defined. It appears that for patients who have undergone complete gross resection, and are surgically staged to show no residual tumor, radiation therapy is not necessary.[5] However, patients who have had only partial resection may benefit from radiation therapy.[9] The usual recommended doses of total radiation are between 1500 and 4000 rads. In late stages of disease, radiation therapy may be used as palliative treatment for symptomatic metastases, in which case 200-300 rads delivered daily for two or three days may be beneficial.[5]

Materials and Methods

From January 1970 through December 1980, 23 patients under the age of 16 years were admitted to the University of Kansas Medical Center with the diagnosis of neuroblastoma. A retrospective study was utilized to determine the following information: age at diagnosis; race and sex; site of tumor; histology of tumor; presence of calcification in the tumor; absolute lymphocyte count; constitutional symptoms; stage at diagnosis; presence of elevated catecholamine metabolites at diagnosis; treatment of the tumor; and survival of the patients. This data was collected and compiled to determine its relationship with existing information and to determine the efficacy of treatment at the Medical Center.

Results and Discussion

Of the 23 patients seen at UKSM during the ten year period, there were a total of 14 males (61%) and nine females (39%). Four of the 23 were black (17%) and 19 were white (83%). Twenty-six per cent of patients were diagnosed before 1 year of age, 50 per cent were diagnosed before 2 years of age, and 70 per cent were diagnosed before 4 years of age. Neuroblastoma in 20 (87%) patients was of

TABLE II
PRESENTING SYMPTOMS

Symptoms at Dx	*# of Patients*	*% of Total Population*
Back/abdominal pain	7	30
Abdominal mass	9	39
Skin flushing	1	4.4
Vomiting/diarrhea	3	13
Anorexia	4	17.4
Intestinal obstruction by tumor mass	1	4.4
Fatigue/lethargy	4	17.4
Elevated blood pressure	1	4.4

abdominal origin and 11 (55%) of these were primary tumors from the adrenal gland. Calcification was present at diagnosis in 78 per cent of the tumors. Twenty-one of 23 patients were checked for elevation of catecholamine metabolites, primarily VMA, and 13 (62%) demonstrated elevated levels.

The most common symptoms at presentation were abdominal mass (39%) and abdominal pain (30%). Other symptoms are listed in *Table II*.

Absolute lymphocyte count (ALC) was checked at diagnosis. Thirteen of the 22 patients checked had an ALC greater than 3000 mm^3.

Therapy for patients with neuroblastoma varied from no therapy to chemotherapy and radiation therapy. The most common agents used were vincristine and cytoxan. Aggressive multi-agent chemotherapy was employed for extensive disease.

Of the 23 patients, four (17%) received radiation therapy only. Two were stage I, and one each stage II and IV-S. All four (100%) are still living. Only one of 23 received chemotherapy only; this patient was a stage IV-S and is still living. Seventeen (74%) of the 23 patients received both radiation therapy and chemotherapy. Of these, one was stage II, four were stage III, seven were stage IV, and five were stage IV-S. Only two patients, both with stage IV-S, are currently living. One patient, stage IV-S, received no therapy, and that patient is still living.

The information gathered for the last 10 years here at UKSM revealed an overall survival rate of 35 per cent. This is comparable to 31 per cent survival rate seen in other studies. Median duration of survival was 106 months, with a range of 71-141 months.

Of the patients diagnosed with neuroblastoma in this series, 87% per cent presented with a primary tumor of abdominal origin. This is considerably higher than in previous studies.[2] Also, 55 per cent of these tumors were of adrenal origin compared with an expected value of 27 per cent.[2] Calcification of the tumor was found to be 78 per cent, compared to a normal value of 50 per cent. VMAs were elevated in 68 per cent of patients compared with the suggested 80 per cent at diagnosis. The small number of patients in the study, however, make it difficult to correlate findings with the expected values.

The absolute lymphocyte count (ALC) indicated a possible prognostic significance. Of the 22 patients diagnosed and checked for ALC, only 13 had an ALC greater than 3000 mm^3, and 54 per cent of these are still living.

Age at diagnosis *vs* prognosis is consistent with previous findings in that there was a definite inverse correlation between age and prognosis. Stage I, II, and IV-S patients had a good prognosis. Stage III and IV had no survivors in this study indicating a poor prognosis *(Table III)*.

The effectiveness of chemotherapy in conjunction with radiation therapy in this study did not correlate well with outcome, considering that 17 patients received both types of therapy with only two surviving for an extended period of time.

(Continued on page 396)

TABLE III
SURVIVAL IN RELATION TO AGE & STAGE AT DIAGNOSIS

Stage at Dx	*Total # of Patients*	*Age of Patient* <1	*Age of Patient* >1	*# Living*	*% Living Disease Free*	*Duration of Survival for Those Not Living*
I	2	1	1	2	100	—
II	2	0	2	1	50	11 months
III	4	1	3	0	0	26 months
IV	7	1	6	0	0	14.75 months
IV-S	8	3	5	5	62.5	10 months
TOTAL	23	6	17	8	35	—

Drug Resistant Tuberculosis

Determination of Effective Drug Therapy for the Indochinese Population in Kansas and Sedgwick County

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Overall *in vitro* drug resistance was examined in the newly immigrated Indochinese population diagnosed with tuberculosis in Kansas and Sedgwick County between 1977 and 1982. Results of the study indicate that a reasonable drug combination for initial treatment in this community could include isoniazid, ethambutol, and rifampin.

THE NEED FOR susceptibility studies in choosing the most appropriate drug regimen in the treatment of disease caused by Mycobacteria tuberculosis has been a point of interest for clinicians for many years. One primary indication is a history of prior antituberculous drug treatment.

Many cases of tuberculosis present without a history of prior drug treatment, and resistance is presumably acquired from existing drug resistant organisms already in the community. The prevalence of this form of resistance is often said to reflect the quality or success of antituberculous chemotherapy in a particular community. The changing patterns and rates of drug resistance during the last decade have established the need to use susceptibility testing routinely.

The arrival of new immigrant populations thought to harbor higher rates of tuberculosis in general, and resistant organisms in particular, has been presumed to be the cause of increasing resistance rates. Recent reports of resistance patterns in the Indochinese refugee population reveals overall resistance to occur in one-third of the population.[1]

The importance of identifying "local patterns" of drug resistance in a particular community, rather than relying on national averages, however, has been borne out by a United States Public Health Service cooperative study.[2] Race or ethnic group of origin cannot be used as the sole predictor of drug resistance in a specific geographic location.

With the increasing use of multiple and short course drug regimens,[3] the importance of identifying rates of resistance in each first-line and some second-line drugs has become apparent. Knowledge of previous treatment can be used by clinicians in planning future retreatment schedules until drug sensitivities are obtained.[4]

Materials and Methods

The number of reported Mycobacteria tuberculosis cases in the newly immigrated Indochinese population of Kansas between 1977-1982 was obtained from state public health records of the Kansas Department of Health & Environment. These were cases with positive bacteriologic specimens as reported by the Tuberculosis Section of the Kansas State Public Health Laboratory. Results of drug susceptibility testing for each reported "positive" case were also obtained.

Through review of foreign language medical records taken at the Wichita Regional Chest Clinic, all Indochinese cases reported positive between 1977-1982 were reviewed to obtain a history of prior antituberculous drug treatment. A similar review of all non-Indochinese index cases reported between 1981-1982 in Sedgwick County was also conducted, in order to compare primary drug resistance in both populations.

Primary drug resistance, to one drug or more, was defined as "identification of resistant organisms, *in vitro,* in patients with no prior history of previous drug treatment."[5] The drugs used in the tests were isoniazid (INH), para-aminosalicyclic acid (PAS), streptomycin (SM), ethambutol (EMB), ethionamide (ETH), and rifampin (RIF). Drug susceptibility testing carried out by the state laboratory followed the procedures recommended by the Centers for Disease Control and as outlined by Vestal.[6]

Results

Drug resistance among the Indochinese in Kansas and Sedgwick County are shown in *Table I*. Of the 57 cases having positive sputum cultures confirmed by the state laboratory, 30 (52%) were localized in

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TABLE I
DRUG RESISTANCE IN THE INDOCHINESE POPULATION IN KANSAS AND SEDGWICK COUNTY, 1977-1982

	Total With Positive Bact.	*Sensitive All Drugs*	*Resistant #*	*%*
Kansas*	27	19	8	30
Sedgwick Co.	30	20	10	33
Statewide	57	39	18	31

* Excluding Sedgwick County.

Sedgwick County.

Similar rates of resistance for Kansas and Sedgwick County were observed — 30 per cent for Kansas, 33 per cent for Sedgwick County.

The preponderance of drug resistance was observed in the last two years — 1981-1982 — of this survey. It was found that eight of the total of ten cases showing resistance in Sedgwick County were reported in those two years.

In *Table II*, a review of the 29 non-Indochinese patients reported to have positive bacteriologic results in 1981-1982 in Sedgwick County revealed 12 (41%) to be resistant compared to 38 per cent found in Sedgwick County for Indochinese patients.

In the Indochinese population in Sedgwick County, ten cases of overall resistance were reported between 1977-1982. Seven of these ten had no history of prior drug treatment, and therefore were primary drug resistant. A large proportion — five of these seven — occurred between 1981-1982, raising the percentage of Indochinese with primary drug resistance to a frequency of 24 per cent *(Table III)*.

A comparison of primary resistance can be made during 1981-1982, with the non-Indochinese population. This shows a larger percentage of primary resistance occurring during this time. The characteristics of this group, as determined through review of patient charts, revealed a high degree of poor patient compliance. Existence of associated morbidity — including alcoholism, one case of immunocompromised host, and two reported deaths — were also present in this group.

In the Indochinese population of Sedgwick County, resistance patterns of each drug revealed that 24 per cent were resistant to INH, none to PAS, 19 per cent to SM, none to EMB, 14 per cent to ETA, and none to RIF *(Table IV)*. In comparison with the non-Indochinese patients during the same period of 1981-1982, distinct differences were noted. This group was composed of Whites, Blacks, and Hispanics, and showed increased rates of resistance to PAS, EMB, ETH, and RIF not noted in the Indochinese. Distinctly higher rates of resistance for INH are noted for the Hispanics.

Conclusion

In studies of drug resistance done between 1961-1973 by both the United States Veterans Hospitals, and the Centers for Disease Control, the rate of overall drug resistance has been placed at 4.0-8.0 per cent.[7] These studies involved mostly non-Hispanic and Asian populations.

In four independent surveys reviewing drug-resistant tuberculosis in the Indochinese population, summarized by the Centers for Disease Control in 1981, it was found that roughly one-third of the population had drug-resistant organisms identified. This is comparable to the 31 per cent frequency of resistance found in Kansas (including Sedgwick County) between 1977-1982 and found in Sedgwick County during this period.

One important result was the high frequency of overall drug resistance of 41 per cent found in the non-Indochinese population in Sedgwick County between 1981-1982. In comparison with the frequency of 30 per cent found in Kansas, the resistance rate for Sedgwick County ($P<0.05$) was not significantly

TABLE II
NUMBER OF POSITIVE BACTERIOLOGY AND PERCENT RESISTANT CASES IN KANSAS AND SEDGWICK COUNTY, 1981-82

	Indochinese			*Non-Indochinese*		
	Total # Positive	*# Cases*	*% Resist.*	*Total # Positive*	*# Cases*	*% Resist.*
Kansas	10	4	40	113	34*	30
Sedgwick Co.	21	8	38	29	12*	41

* Difference was not significant ($P<0.05$) in comparing percentage of resistance for Kansas and Sedgwick County.

TABLE III
NUMBER OF POSITIVE BACTERIOLOGY AND PRIMARY RESISTANT CASES IN SEDGWICK COUNTY, 1981-82

	Indochinese			*Non-Indochinese*		
	Total # Positive	*# Cases*	*% Primary Resist.*	*Total # Positive*	*# Cases*	*% Primary Resist.*
Sedgwick County	21	5*	24	30	9*	30

* Difference was not significant ($P<0.05$) in percentage of primary resistance for Sedgwick County.

different from that for the rest of Kansas, for reasons not determined by this study.

Recent studies have reported findings that have varied in their report of primary resistance rates. In a Canadian cooperative study of Asian immigrants during a 12-year period, this was found to be 11.7 per cent.[8] A more recent study by the Centers for Disease Control of 3,100 cases of Asian patients, seen between 1975-1977, reported a rate closer to 20.7 per cent.

Comparable rates of 23 per cent were found in the Indochinese population of Sedgwick County. However, the non-Indochinese population again showed proportionally higher rates of 30 per cent during the same time period.

Primary resistance patterns are high for both the Indochinese and non-Indochinese populations of Sedgwick County. There are no significant differences between these two frequencies of resistance ($P<0.05$). Some causes of these patterns can be postulated and may differ. The Indochinese group involves a high rate of primary resistance already in existence in that population. In the non-Indochinese population several factors may be involved including: poor compliance; inadequate treatment; or treatment with the wrong drug.

The pattern of drug-resistance for both the Indochinese and non-Indochinese populations in Sedgwick County appear to be similar. However, the former group has a higher frequency of INH resistance due to the frequent use of this drug in Asian countries.

A recommended regime of triple drug therapy, initially, in areas with a high prevalence of drug resistance has been advocated by the American Thoracic Society. This includes the addition of two new drugs together with INH, initially, while awaiting susceptibility studies. Once susceptibility resistance patterns are known there is a choice between two approaches: (1) Add two new drugs together with INH; or (2) Add three new drugs with INH, in cases of life threatening infections.[9]

Resistance to ethambutol and rifampin was rarely found in Sedgwick County between 1981-1982. The only case of resistance to EMB, PAS, or RIF was found in a non-compliant alcoholic. Considering this low frequency of resistance, a reasonable drug combination to initiate treatment in this community can include INH, EMB, and RIF.

In vitro susceptibility testing has enabled us to identify resistance patterns in populations within our community. This can be an important tool in monitoring resistance in newly arriving immigrant populations in the future. *(Continued on page 413)*

TABLE IV
IN VITRO ANTITUBERCULOSIS DRUG SUSCEPTIBILITY PATTERN IN SEDGWICK COUNTY, 1981-82

		≥1 Drug	*INH*	*PAS*	*SM*	*EMB*	*ETH*	*RIF*
Indochinese (# positive bact. 21)	# Resistant	8	5	0	4	0	3	0
	% Resistant	38	24	0	19	0	14	0
Non-Indochinese (# positive bact. 29)	# Resistant	12	5	1	3	1	10	1
	% Resistant	40	16	3	10	3	33	3



Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Asbestosis and Asbestos-Related Disease

DIANA S. DARK, M.D.* *and* SUSAN K. PINGLETON, M.D.,† *Kansas City, Kansas*

Asbestos is one of the world's most useful minerals, used today in the manufacture of more than three thousand products. Although asbestos was in use as early as 2500 B.C., wide-scale use did not begin until the industrial revolution when insulation was needed for the steam engine. Since the turn of the century, approximately 3×10^7 tons of asbestos have been used in the United States.[1]

Deposits of asbestos are ubiquitous in the earth's crust in many geographic formations. Currently, six naturally occurring fibrous silicate materials are considered by industrial and governmental groups as asbestos.[2] On the basis of mineralogic features, they are divided into two groups, serpentine and amphibole. Chrysotile, composed of pliable, curly fibers, is the only commercially available mineral of the serpentine group. The fibrillar subunits of chrysotile are arranged in pseudohexagonal arrays composed of silicon oxide sheets formed into scroll-like structures. The three major amphiboles available are crocidolite, anthophyllite, and amosite. This group is composed of straight, rodlike fibers consisting of double chains of tetrahedral groups having a basic silicon oxide composition and linked by one or more cations.

The U.S.S.R. and Peoples Republic of China are the major suppliers of asbestos worldwide although Canada and South Africa also contribute heavily. Chrysotile currently accounts for more than 90 per cent of the total asbestos marketed worldwide. Only a few relatively small mines are active in the United States, although serpentine and amphibole minerals are distributed widely in the geographic strata.[1]

Advantages of asbestos include its strength, moldability, and fire and corrosion resistance; it is also less expensive than most man-made substitutes. The manufacture of asbestos-cement products consumes the largest share of asbestos fiber with the floor tiling industry consuming the next largest quantity. The fiber is used widely for insulation and fireproofing, and asbestos textiles are used in fire-protective clothing.

Asbestosis

Of the many disease processes associated with asbestos exposure, the most common is asbestosis *(Table I)*. Asbestosis is a bilateral, diffuse interstitial pulmonary fibrosis which is associated with prolonged, heavy exposure to asbestos. The pathogenesis is not completely understood but the initial event appears to be phagocytosis of the asbestos fiber by a macrophage.[1] This appears to be a prerequisite for the development of pulmonary fibrosis. Alveolar macrophages contain high levels of lysosomal hydrolases and proteases, including elastase. During phagocytosis, lysosomal enzymes and soluble immune modulators are released outside the macrophage; they may cause widespread tissue destruction, granuloma formation, and pulmonary fibrosis. When engulfed by macrophages, the fibers may become incorporated within a mucopolysaccharide and iron matrix forming coated fibers, known as asbestos or ferruginous bodies.

In asbestosis, the number of *uncoated* fibers in the lung greatly exceeds the number of asbestos bodies in the tissue. It is not known why some fibers are coated and others not. Attempts to correlate the extent of disease with either the number of asbestos

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TABLE I
ASBESTOSIS

- Bilateral, diffuse interstitial pulmonary fibrosis.
- Associated with prolonged, heavy asbestos exposure.
- Associated with restrictive pulmonary function tests.
- Diagnosed clinically or by lung biopsy.
- No specific therapy is known.

bodies or overall content of fibers in the lungs have been inconclusive, although it is generally agreed that the uncoated fibers are responsible for more damage. The consequences of phagocytosis of asbestos dust by the macrophage appear to reflect the intrinsic cytotoxic potential of the type of asbestos concerned.[3] Chrysotile has shown significantly greater *in vitro* cytotoxicity than either crocidolite or amosite. Asbestos also activates complement by the alternative pathway, a reaction that may be expected to result in accumulation of leukocytes in the tissue and the release of lysosomal enzymes.[1]

The primary clinical features of asbestosis are shortness of breath and gradually worsening dyspnea on exertion. In later stages the patient may develop a non-productive cough. Physical signs include equilateral impairment of expansion affecting the lower chest wall and bilateral lower lobe crepitations which increase with progression of the disease. The earliest abnormalities of pulmonary function are decreased compliance and diffusing capacity; these may even precede symptoms. There is usually widening of the alveolar-arterial gradient and, eventually, restrictive changes with decreased total lung capacity and functional residual capacity develop. Radiographically, asbestosis appears as progressive linear opacities most prominent in the lower lobes.

Diagnosis of asbestosis can be made either clinically, after rigorous exclusion of similar disease, or by lung biopsy. Indications for lung biopsy are (1) severe constitutional symptoms, (2) atypical development, distribution, or progression of radiologic appearance, or (3) the presence of a disorder associated with interstitial pneumonitis. Microscopic examination of biopsy specimens may reveal intra-alveolar desquamation of type 1 cells, collagenous fibrosis, and patchy bronchiolitis obliterans. Condensation of the elastic framework of alveolar walls may be present; asbestos bodies are usually present and often occur in clusters within and adjacent to areas of fibrosis.[4] These findings are characteristic but not diagnostic of asbestosis.

Asbestosis is a disease that tends to progress slowly even many years after exposure has ended; progression may cease at any stage. Once the diagnosis is established, the patient should be removed from further exposure. No specific therapy for this disease exists.

Pleural Plaques

Pleural plaques are also associated with asbestos exposure. These plaques are made of hyalinized fibrous tissue and usually located on the parietal pleura of the thorax, diaphragm, mediastinum, or pericardium. Occurrence of plaques seems to correlate with duration and intensity of asbestos exposure and may be found in the absence of parenchymal pulmonary disease. The configuration of the plaques is highly variable; on the chest wall they usually follow the contour of the rib, and on the diaphragm they tend to be either disk-shaped or geometrically shaped and have a nodular surface.

Microscopically, the plaques consist almost entirely of avascular, acellular laminated collagen fibers arranged parallel to the surface, with hyaline changes. Dystrophic calcification is present in central areas where collagen has undergone degeneration. The most external parts of the plaques show fibroblastic activity with collections of lymphocytes and plasma cells, indicating that plaques are of extrapleural, rather than pleural, origin. Over time, the lesions become calcified and are seen easily by radiographs[1] *(Figure 1)*. The pathogenesis of plaque development is not completely understood, but the



Figure 1. Chest roentgenograph showing pleural plaques (arrows).

appearance of plaques is particularly associated with exposure to anthophyllite. Suggested etiologies are penetration of sharp fibers into the pulmonary pleura during respiration where they pass directly into the parietal layers or direct access to the pleura via lymphatic vessels.[1] The patient usually remains asymptomatic unless the calcified plaques are very extensive and cause mild restriction of chest wall movement.

Radiographically, calcified plaques have an irregular, unevenly dense pattern which has neither segmental nor lobar distribution, thus indicating the extrapulmonary site. Diagnosis is made either radiographically or at autopsy. The plaques have no effect on life expectancy and are not known to give rise to any complications; no treatment is indicated.

Malignant Mesothelioma

Malignant mesothelioma is a tumor of the cells of mesodermal origin lining the pleural, pericardial, or peritoneal cavities. The precise incidence of mesothelioma is unknown, but its relationship to asbestos exposure was confirmed among women filling gas mask filters during World War II. The risk of developing mesotheliomas is apparently dose-related although the tumors have occurred in persons with trivial or indirect exposure.[1] Pathogenesis is not clear but seems to be related primarily to the length and diameter of the fibers. These tumors are most commonly located on pleural or peritoneal surfaces.

Initial symptoms of pleural tumors include dyspnea, chest pain, or both, occurring in greater than 90 per cent of cases.[5] Both worsen gradually as the disease progresses. As with other malignancies, malaise and weight loss occur later. Peritoneal tumors are more likely to be silent until the disease is extensive. The patient may complain of vague intermittent abdominal pain, anorexia, and weight loss.[6]

Signs of pleural mesothelioma include a painful chest wall and unilateral pleural effusion. Occasionally the patient will present with an ipsilateral pneumothorax. At thoracentesis the pleural fluid is usually sterile, clear and yellow, but may be frankly hemorrhagic, gelatinous, and difficult to aspirate. Cytological examination may not be helpful because of the difficulty in differentiating malignant mesothelial cells from normal cells; normal mesothelial cells in any pleural effusion can assume a bizarre appearance.[7, 8]

Signs of peritoneal mesothelioma may be absent initially but ascites soon becomes evident. The abdomen may be distended and firm; masses become palpable. Other than a possible thrombocytosis,[6] routine laboratory data are usually not remarkable in either pleural or peritoneal mesothelioma.

Pleural mesotheliomas are manifest radiographically as irregular, lobulated, well-demarcated, protuberant opacities that line part of the chest wall and may extend into the lung fields *(Figure 2)*. A unilateral pleural effusion may be present; bilateral calcified pleural plaques and basal interstitial fibrosis may be present but are uncommon findings. Radiographs are usually not of benefit in diagnosing peritoneal mesothelioma, except to show the frequently associated pleural plaques.

Biopsy may be necessary to exclude more treatable types of tumors but can be difficult to interpret. Rather large amounts of tumor often are needed to confirm the diagnosis. In one study, the correct diagnosis was made in 26 per cent of needle biopsies, 38 per cent done by thoracotomy, and 70 per cent of surgical excisions.[4] Any procedure must be carefully considered because tumors are likely to advance along needle tracks or surgical incisions.

Prognosis is extremely poor and the disease is uniformly fatal. With pleural disease the average life expectancy is 16 months after diagnosis; with peri-

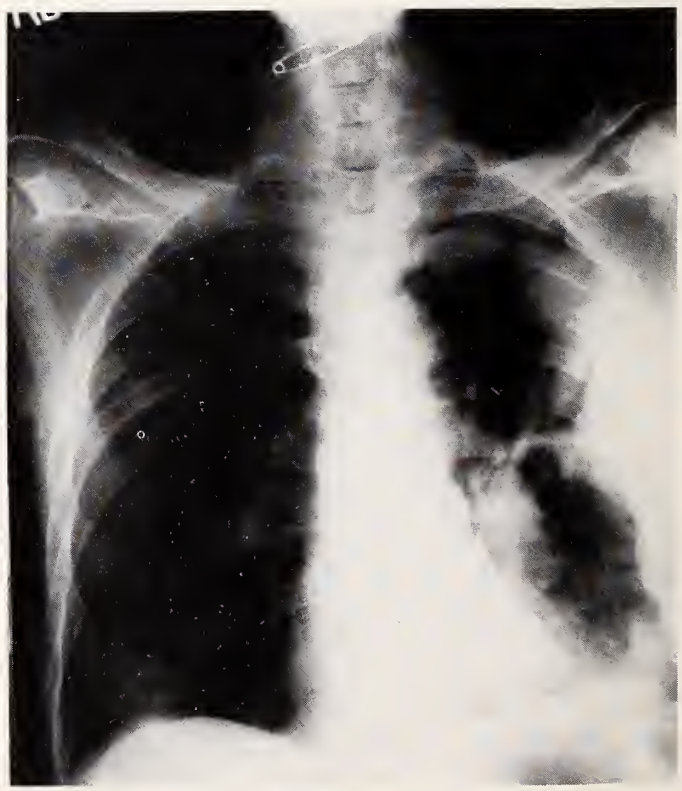


Figure 2. Chest roentgenograph showing left-sided pleural-based density diagnosed as mesothelioma.

toneal mesothelioma the average expectancy is nine months.[6] Treatment is essentially palliative. Therapeutic thoracenteses can be helpful but become increasingly difficult to perform as the tumor thickens and hardens. Sclerosing agents have proved to be of some benefit. Radical surgery is rarely, if ever, curative. Partial decortication and pleurectomy with good palliation of pain can improve the patient's quality of life. Chemotherapeutic agents have met with poor to marginal success. Doxirubicin has been the most extensively tested with disappointing results overall. Multiple combinations have been tried with little success, although the doxirubicin/5-azacitidine combination has had limited success. In a recent study of 69 patients, there were four complete responders, all of whom began therapy with extensive, unresectable or metastatic disease. These four patients lived 27 to 76 months from the onset of chemotherapy.[6]

Benign Asbestos Pleural Effusion

Another disease process associated with asbestos exposure is benign asbestos pleural effusion. Development of the effusion may be acute, with fever, chest pain, leukocytosis and increased erythrocyte sedimentation rate, or chronic, with few or no symptoms. There is no correlation between the occurrence of the effusion and the duration of exposure to asbestos. Effusions may be single or recurrent, uni- or bilateral. Pleural fluid is often bloody and may contain asbestos fibers or bodies. The following four criteria define this disease: (1) exposure to asbestos; (2) confirmation of effusion by x-ray or thoracentesis; (3) rigorous exclusion of other diseases associated with pleural effusion; and (4) no malignant tumor within three years. This is obviously a retrospective diagnosis and no therapy is indicated.[9]

Bronchogenic Carcinoma

Epidemiologic studies have documented an association between bronchogenic carcinoma and occupational exposure to asbestos.[10] Prevalence is higher in persons working with finished products (such as insulators) than in miners and millers. The severity of any associated pulmonary parenchymal fibrosis (asbestosis) correlates with an increase in the number of neoplasms. The incidence of tumors also is increased in asbestos workers who lack radiologic evidence of asbestosis. Adenocarcinomas are the most predominant tumors. The lesions tend to occur more frequently in the lower lobes in conjunction with more severe degrees of fibrosis.[1] Whereas there is a slight increase in the prevalence of lung cancer among non-smokers with asbestos exposure, heavy smokers with exposure show an 80-to-90 fold greater predisposition to bronchogenic carcinoma over non-smoking, non-exposed persons.[11] Thus, the synergism between asbestos and smoking appears to be multiplicative rather than additive. The precise role of asbestos is not known. The fibers may serve as a physical carrier of the carcinogens in the cigarette smoke to the basal layer. Another hypothesis is that the alveolar macrophage, which phagocytizes asbestos in the airways, may convert the polycyclic hydrocarbons to active metabolites. Recent projections suggest that approximately 1 per cent of lung cancer in the rest of the century will be due to asbestos. Such projections emphasize that cigarette smoking continues to be the most common cause of lung cancer.

Other malignancies thought to be associated with asbestos exposure include upper and lower gastrointestinal tract tumors, oropharyngeal, esophageal, pancreatic, and renal tumors. The pathogenesis of the increased prevalence of these associated carcinomas is unknown.

TABLE II
DISEASES ASSOCIATED WITH ASBESTOS EXPOSURE

Disease	*Appropriate Therapy*
Asbestosis	none known
Pleural plaques	none indicated
Malignant mesothelioma	palliation; consider chemotherapy
Benign asbestos pleural effusion	none known
Bronchogenic carcinoma	surgery; radiation therapy; chemotherapy

Summary

Asbestos is a mineral present in many products to which thousands of people are exposed every day. The exposure is often hidden or forgotten, leaving people unaware and unprotected. Disease processes caused by and associated with asbestos exposure are being recognized with increasing frequency *(Table II);* because of the long latent period many feel that there will be an outbreak of asbestos-related disease later in this century. Because of this and of increasing public concern, physicians need to be cognizant of asbestos exposure and its implications, and to be alert for possible asbestos-related disease.

Self-Assessment Questions

Directions: Choose the single best answer to each question.

1. Major suppliers of asbestos include:
 a. United States
 b. Spain
 c. U.S.S.R.
 d. Mexico
 e. all of the above
2. Uses of asbestos include:
 a. insulation
 b. fire-protective clothing
 c. floor tiling
 d. brake and clutch linings
 e. all of the above
3. The earliest abnormality of pulmonary function in asbestosis is:
 a. increased residual volume and total lung capacity
 b. decreased FEV_1
 c. decreased FVC
 d. decreased diffusing capacity
 e. decreased total lung capacity
4. The appearance of pleural plaques is especially associated with:
 a. chrysotile
 b. crocidolite
 c. anthophyllite
 d. amosite
5. True statements regarding malignant mesothelioma are:
 a. The tumors may be located in the pleural, peritoneal, or, most commonly, pericardial cavities.
 b. the tumors are usually asymptomatic and discovered by routine x-ray or at autopsy.
 c. The pathogenesis seems to be related primarily to the length and diameter of the fibers.
 d. Diagnosis can always be made by cytological examination of pleural fluid.
 e. All of the above.

(Answers on page 413)

Join KaMPAC

Neuroblastoma in Children

(Continued from page 388)

Conclusion

From this study, as was evident in previous studies, there is a definite correlation between prognosis and stage of the disease and age of the patient at diagnosis. Stage I, II, and IV-S have a much better prognosis and children under 1 year of age do better than older children.

Surgery with or without radiation therapy seems to be the best treatment modality at this time. Radiation therapy should be given to patients with incomplete resection of the primary tumor. The effectiveness of chemotherapy has been debated in neuroblastoma. Various combinations of chemotherapy from mild to very aggressive have been proposed to be effective. However, consistent effectiveness remains questionable. Recently, Hayes *et al.*[10] have attempted to correlate clinical response to chemotherapy with *in vitro* cell cycle kinetics and to develop new potentially effective chemotherapeutic regimens. Patients with disseminated disease showed good response if cyclophosphamide was given daily for seven days, followed by adriamycin on the eighth day. A combination of VM-26 and Ara-C given 48 hours apart also demonstrated marked effectiveness based on *in vitro* cell kinetic studies. These regimens currently are being tried in a cooperative group setting.

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Drug INFORMATION

JOYCE GENERALI, R.Ph., *Editor*

Drug-Induced Vitamin Deficiencies

Question: Will you provide information regarding drug-induced vitamin deficiencies?

Answer: Drugs may induce vitamin deficiencies by a direct or indirect effect of the drug on vitamin absorption, utilization, metabolism, or excretion. Some agents, such as those causing appetite suppression or taste disturbances, may lead to a reduction in normal body vitamin stores due to the potential lack of consumption. Specific drugs reported to induce vitamin deficiencies are listed below.

- *Anticonvulsants* — especially barbiturates and hydantoins — may decrease the absorption of vitamin B_{12} and folate by interference with gastrointestinal absorption and inhibition of intestinal absorptive enzymes.[1-3] Anticonvulsants may also accelerate the metabolism of vitamin D by liver enzyme induction.[4]
- *Cholestyramine* has been associated with vitamin K deficiency during long term therapy.[5] Other potential vitamin deficiencies induced by cholestyramine include vitamins A, D, and B_{12}.[6] The mechanism of these interactions is believed to involve binding of vitamins by cholestyramine.
- *Colchicine* administered orally decreased the absorption of ^{57}Co-labeled vitamin B_{12} in 18 of 19 subjects studied.[7] In daily doses of 1.9-3.6 mg, colchicine decreased urinary excretion and increased fecal excretion of vitamin B_{12}. Colchicine is thought to alter the function of the ileal mucosa, thus decreasing absorption of vitamin B_{12}.
- *Folic acid antagonists* (including methotrexate, triamterene, sulfonamides, and trimethoprim) inhibit the enzyme dihydrofolate reductase leading to decreased utilization of folic acid.[8, 9]
- *Isoniazid* is reported to combine with pyridoxal or pyridoxal phosphate (active forms of vitamin B_6) forming an inactive hydrazide complex.[9]
- *Methyldopa*-induced vitamin B_{12} deficiency is suggested in at least one case report.[10] This deficiency was reported as part of a malabsorption syndrome. An autoimmune mechanism may be responsible for this interaction; however, data are not available to support this theory.
- *Mineral oil* is thought to decrease the absorption of carotene (precursor of Vitamin A), and vitamins D, E, and K.[11, 12] This may be due to adsorption of vitamins or changes in the gastrointestinal tract caused by mineral oil.
- *Neomycin* decreases carotene plasma levels and the urinary excretion of ^{60}Co-labeled vitamin B_{12}.[13] The mechanism of these interactions may involve changes in the gastrointestinal normal flora, hypermotility of the bowel, inhibition of intestinal absorptive enzymes, or changes in gastrointestinal cells causing physical blockage of absorption.
- *Oral contraceptives* have been shown to decrease the absorption of polyglutamic folic acid (dietary folate) by approximately 50 per cent.[14] The absorption of monoglutamic folic acid (vitamin preparations) does not appear to be affected, suggesting the mechanism of malabsorption involves inhibition of intestinal absorptive enzymes.
- *Para-aminosalicylic acid (PAS)* is reported to decrease the absorption of vitamin B_{12}.[15, 16] This interaction is thought to occur after a minimum of two weeks of PAS therapy, and involves inhibition of intestinal absorptive enzymes.
- *Penicillamine* interferes with the metabolism of vitamin B_6.[17] It is suggested that penicillamine, a chelating agent, may chelate vitamin B_6 and increase its excretion.[17, 18]
- *Potassium chloride* (slow release products) may decrease the absorption of vitamin B_{12} due to

acidification of ileal contents.[19] This interaction appears to occur following chronic administration of slow release potassium chloride products.

- *Salicylazosulfapyridine (Azulfidine)* is reported to decrease the absorption of monoglutamic folate in patients with ulcerative colitis and granulomatous colitis.[20] Folate absorption studies performed prior to and during salicylazosulfapyridine therapy confirmed the reduction in folate absorption.

Submitted by
Bruce Scott, R.Ph.

References are available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

The Drug Information Service is maintained by the Department of Pharmacy at the K.U. Medical Center to promote rational drug therapy. The Service is staffed by clinically oriented pharmacists with access to accurate, current, and unbiased information. Information is available free of charge to any health care practitioner involved with patient care. Contact:

Drug Information Service
3930 Cambridge
Kansas City, KS 66103

Telephone (any time): 913-588-2328

Bendectin Discontinued

Bendectin, the controversial antinausea/vomiting medication, will no longer be produced by Merrell Dow Pharmaceuticals.

In announcing their decision, Merrell reiterates its conviction that Bendectin has been thoroughly and adequately tested, and there is no conclusive evidence of a cause and effect relationship between its use and birth defects.

Nevertheless, Merrell feels that the negatives effects of adverse publicity cannot be surmounted, and that the burdens of litigation and publicity — on physicians, pharmacists, and patients, as well as Merrell — make this action necessary.

Merrell states that the decision to cease production of Bendectin was based solely on non-medical factors. The company anticipates that present stocks are adequate for completion of therapy for current patients.

Persons desiring additional information may call 800-543-1970.

The President's Message

The number of physicians in Kansas has been increasing the last few years. An increase of 109 occurred in 1982. The number of underserved counties decreased from 55 to 36 during the past year.

More than 3000 physicians are practicing in Kansas, and nearly 90 per cent are members of the Kansas Medical Society. The increase in numbers has also given Kansas a bigger voice in the American Medical Association; we now have an additional delegate to the AMA.

Exchanging ideas with physicians from other sections of the country allows introspection of the positions and theories that occur in a state. Although much attention is directed to national issues, the implementation of these issues is on a local, state, or regional level.

The next few months will bring much information to you about the new trends in third-party participation, state government involvement, and federal guidelines. Your medical society will continue to be involved in all of these areas with input, interpretation, and innovation of new ideas.



Please contact us about your ideas. We can only serve you better with interaction.

James A. Pearson, M.D.
President



What's In Whose Name?

The vital signs in the *Journal* office are back to normal although there is still a slight elevation of the serum chagrin level. This situation is due to a recent exacerbation of a condition endemic in editorial offices, the typographical error. This attack was manifested by the appearance in the March issue of a ''Current Comment'' entitled ''Munchausen's Syndrome and Other Factitious Disease'' which was heralded by the ''blurb'' line, ''The Great Imposter.'' Since, of course, it should have been ''Impostor,'' we stand convicted on a straightforward *res ipsa loquitur* charge.

Having overlooked the error in the first place, we might have gone on in happy ignorance of the gaffe had we not received indirectly direct word from Dr. James Kalivas of the Department of Dermatology at the Medical Center in Kansas City. (The ''indirectly direct'' refers to the fact that Dr. Kalivas forwarded a righteously indignant letter to a colleague but saw to it we got a copy.) We hope he has been placated by our (still) trusted editorial assistant's generous assumption of guilt — she might at least have diluted it on her own behalf since the error was compounded by the fact that it passed through several hands — and eyes.

It can be noted, however (to dismiss this opening gambit with more attention than it really deserves), that the ''imposter-imposter'' problem has caused trouble in publishing circles on more than one occasion. We recall one discussion in which the author tried to put the matter to rest by concluding that an imposter could only be one who assesses or collects imposts which should win the Driven Snow Award. Still, the Encyclopedia Britannica Dictionary and our old stand-by, Fowler, conclude it is permissible either way (but not eith*or* way). If anyone is really interested, the basic rule is that such agency words of Latin origin take the *-or* while those of English origin take the *-er* (and it shouldn't take you long to find exceptions to *that*). After all this, we make no claim that our usage was top-drawer or other than an error, and we add this final (we hope) *mea culpa* as testimony that we shall continue the sincere but largely futile effort to keep our pages free of such blemishes.

If it appears that we are lacking in regret over this article, we are not, but it is of a personal and quite different order. From our first awareness that this complex of self-destructive entities had been tagged with the title, ''Munchausen's Syndrome,'' we have felt it was not only inappropriate, erroneous, and misleading but an unwarranted affront to a gentleman for whom we have had high regard since about the fourth grade. Let us make it clear that this is no criticism of the authors of this particular offering. They did just as we did — used a generally accepted designation which has now, we fear, become a permanent fixture of medical jargon. We realize our objections will smack of journalistic petulance since we failed to mount any counteraction at the time and raise the point at this late date only for a little midsummer spleen-venting. The gentleman who has become unknowingly identified with the syndrome was, of course, the Baron Munchausen whose facility in spinning tall tales has been equated at some point with the fabrications that mark the *modus operandi* of the factitious disorder patient. It has even been suggested that the Baron's travels as reflected in his stories are comparable to the progression of the ''victim'' of self-induced disease from one medical installation to another. Well, the Baron did do a fair amount of traveling in pursuit of his military duties but the travels incorporated in his tales are part of their happy but imaginary structure and are in no way comparable to the peregrinations of the psychopaths he has been coupled with.

Hieronymus Karl Friedrich, Freiherr von Munchausen of Bodenweder, Hanover, was, it would seem, a *gemütlich* soldierly type, an uncomplicated extrovert whose chief pleasure was sharing a bottle and his stories with his friends. He didn't, as a matter of fact, ''write'' them — the individual credited with

recording the original ones was one Rudolph Erich Raspe, a professor at the University of Kassel. We say "original ones" because the full group of stories as currently published includes others added by subsequent bookseller-publishers who passed the publication along and couldn't resist adding to the fun (all of which was quite permissible, copyright laws being what they were — or weren't). Whatever the Baron's aptitudes, his personality was such as to fix his name to the genre and two centuries of children have happily enjoyed this characteristically human indulgence in the challenge of fantastic thoughts, the elevation of the fib to the socially acceptable level, a sort of joke on grown-ups who stuffily demand the unblemished truth (from others). Since most children have very little success in putting over their transparent tales, it's no wonder they are attracted to the combination of mendacity and impunity which marked the Baron's efforts. (But only children and politicians understand that a thing not being true doesn't necessarily make it a lie.)

So the Baron's purpose and method were antithetical to the psychological aberrations embodied in the individuals with self-induced disorders. There is only the most tenuous generic connection between the prevarications of the two — unless one wants to say that both work toward gaining attention and status for the principals. In fact, one wonders if there has been some semantic confusion between "factitious" and "fictitious" — a sort of subliminal typographical error, shall we say? No, we'll remain obdurate — we won't put the Baron's pleasure-oriented accounts in the same pigeon hole with the ultimately futile efforts of the factitious disorder "victim" to gain credibility with medical attendants.

If a personal name has to be attached to the syndrome (and that long-established practice annoys us too but there is no point in tilting at *that* windmill at the moment and there is a limit to our petulance), we would nominate the original recorder of the Baron's adventures, the aforementioned Raspe. His duties at the University of Kassel included being curator of the museum. When he was commissioned by the landgrave of Hesse to go to Italy to obtain some curios for the museum there, he was so successful that some found their way into his pocket for his personal gain, a no-no in the opinion of his sponsors, and he had to light out for England. There he came under the patronage of one Sir John Sinclair, but his acquisitive habits wore out his welcome there as well. With a seemingly self-destructive persistence worthy of the factitious patient, he conned the gentleman into believing his estate had valuable veins of precious minerals — until it was discovered that Herr Raspe had done a little "salting." Again, he had to take off hurriedly, this time to Ireland where he either mended his ways or found more tolerant friends. Not only does he seem more suitable for the role of association with such a complex of pathologies but — the Raspe Syndrome — doesn't that have a more factitious sound?

But we can't depart the scene without bringing up one more complaint. (Why doesn't he move off that burr instead of just sitting there howling?) And we acknowledge there are some who will think there are greater problems confronting us — the Middle East, Central America, cost containment — but these must wait their turn. Now, we grant we won't get far in divorcing the considerable number of syndromes from their proper names in favor of more clinical designations, but can't we at least get rid of the possessive? The only success in this direction has been limited and mainly applied to new syndromes, but the older ones are stubborn (as are the older ones in any context). After all, the Baron was not afflicted with any of the manifestations of the problem so why is it implied that it is characteristic of him? He had no proprietary interest in them although he might have enjoyed — even admired — some of the stories the patients develop. But most obviously, he was not a physician and played no role in the clinical efforts to define these aberrations as a syndrome. We admit, were he to know of the current situation, he might even enjoy the dubious honor of the connection but that wouldn't change our mind. When one takes up a cause in another's behalf, one can't be too concerned with the subject's feelings — that's the first lesson reformers learn. We must be prepared to sacrifice if we are to exorcise the tyranny of improper traditional terminology — including the nonsensical *'s*.

But we just had a sudden thought. We're not familiar with the 18th Century Hanoverian bureaucracy, but such principalities usually relied upon various ones of the titled citizenry to collect taxes (one of the perks, as it were). It just may be that Baron Munchausen was, in fact, an imposter — oh, no, let's not get started on that again!

Gute Nacht, Hieronymus, wo immer Sie seien. Jedenfalls versuchten wir. — D.E.G.

Attitudes in the U.S. Toward Alcohol and Other Mood Altering Drugs

GEORGE M. PENN, M.D.

THE UNITED STATES is confronted with a crisis of epidemic proportion: dependence upon alcohol and other mood altering drugs, including prescription drugs. Our increasing promotion of and reliance upon these substances are destroying the lives of millions of people — our son, daughter, mother, father, husband, wife, neighbor, friend. Each of us has a personal responsibility to become more concerned, more informed, more vocal, and to take concerted action to stop this epidemic.

We need, first of all, to understand the type of thinking that characterizes our society today and that influences the shaping and developing of public policy. It should be apparent that we are no longer a Judeo-Christian society; we have become increasingly secular and materialistic. Secularism is the hallmark of our society and it is the legacy that we are leaving to our children. In today's society we allow those who are in power to decide what we shall hear, read and see and, it follows, what we shall think and what we shall value.

What qualities characterize 20th century society?

Is there something fundamentally wrong with our world view?

Who is influencing this view?

How does such a world view help to explain our current epidemic drug crisis?

Are we helpless to do anything about it?

We live in a crisis of intellectual, moral, and spiritual dimension; a crisis of a scale and urgency unprecedented in recorded human history. Fritjof Capra, author of *The Turning Point,* says:

> It is a striking sign of our times that people who are supposed to be experts in various fields can no longer deal with the urgent problems that have arisen in their area of expertise. Economists are unable to understand inflation; oncologists are confused about the causes of cancer; psychiatrists are mystified by schizophrenia; police are helpless in the face of rising crime.[1]

Educators are frustrated — babysitting instead of teaching; chemical dependence experts are puzzled by the increase in alcoholism and other drugs of dependence, and the list goes on. In the United States, it has been traditional for presidents to turn to academic people for counsel, either directly or through "brain trusts" and "think tanks" set up explicitly to advise government on various policy matters. This intellectual elite has formulated the "mainstream academic view" and generally agreed on the basic conceptual framework underlying its advice. Today this consensus no longer exists. In 1979 the *Washington Post* ran a story under the heading, "The Cupboard of Ideas Is Bare," a story in which prominent thinkers admitted they were unable to solve the nation's most urgent policy problems. According to the *Post,* "Talks with noted intellectuals in Cambridge, Massachusetts and New York, in fact, not only confirm that the mainstream of ideas has split into dozens of rivulets, but that in some areas it has dried up altogether." One of the academics interviewed was Irving Kristol, Henry R. Luce Professor of Urban Values at New York University, who said that he was resigning his chair because "I don't have anything to say anymore. I don't think anyone does. When a problem becomes too difficult, you lose interest." In an effort to explain the sources of their confusion or retreat, the intellectuals cited "new circumstances" or "the course of human events" — Viet Nam, Watergate, and the persistence of slums, poverty, and crime. None of them, however, identified the real problem that underlies our crisis of ideas: the fact that most academics — those to whom we turn for answers — subscribe to a narrow perception of reality which is inadequate for dealing with the major problems of our times.[1] No one suggested, at least not seriously, that the problem is that we live in a secular, materialistic society. A society that is bounded by the here and now. A society in which man is the ultimate being and nothing transcends him. A society that denies the existence of eternal values.

There are those, however, who are beginning to question this view of life. They are asking, and rightly so, "What in the world is real?"

People are beginning to realize that something fundamental has changed. Law and government today no longer provide a foundation of justice and

This article has been adapted from the text of a major address by Dr. Penn to the drug abuse conference, "An American Crisis: Wellness *vs* Abuse," April 13-14, 1983, in Salina.

morality, but have become the means of licensing moral perversions of all kinds. Education has become the enemy of religious truth and values. And the media have provided the means for propagating the change.

"There are times when anyone with a shred or moral principle should be profoundly angry. We live in such times."[2]

For example, is there a day that goes by in which the use of drugs is not advertised and glamorized? Our children are receiving a drug education program virtually every time they read — if they can — or watch television.

A major issue for the public and for policy makers is not only the information and its potential effects which we acquire from newspapers and television, but even more so the information that we do *not* get.

For example, the near total absence of stories concerning advancement in treatment and prevention of alcohol and drug dependence, suggest either that we have been remiss in publicizing our good side or exceptionally negligent in highlighting our bad side. More public information concerning positive rehabilitation programs is clearly needed lest the public infer that drug dependence is hopelessly chronic and impossible to treat.

The kind of material presented in newspapers and other mass media must be judged against the motivation policies and opinions of editors and publishers.

There is little doubt that the media of mass communication has great influence on all of us. Has this influential power been calculatingly planned? Have we been "programmed" to think what the media would have us think and to value what it would have us value?

In 1951, Marshall McLuhan wrote:

> Ours is the first age in which many thousands of the best-trained individual minds have made it a full-time business to get inside the public mind. To get inside in order to manipulate, exploit, control, is the object now, and to generate heat not light, is the intention. To keep everybody in the helpless state engendered by remaining in a prolonged mental rut is the effect of many ads and much entertainment.
>
> A huge passivity has settled on industrial society. For people carried about in mechanical vehicles, earning their living by waiting on machines, listening much of the waking day to canned music, watching packaged movie entertainment and encapsulated news, for such people it would require an exceptional degree of awareness and an especial heroism of effort to be anything but supine consumers of processed goods. Society begins to take on the character of the kept woman whose role is expected to be submission and luxurious passivity. Each day brings its additions of silk, trinkets, shiny gadgets, new pleasure techniques, and new pills for pep and painlessness.
>
> *Vogue* is a perfect expression of this state of mind and body. It often plans whole months for its readers, giving exact instructions for what to see, say, eat, and read or wear for each hour of the day. It deals with its readers as a sultan with his harem, as *Time* magazine deals with its readers as a sultan with his eunachs. *Vogue* and *Time,* like the radio, are major political forces shepherding their flocks along the paths of comfort and thrills.[3]

Why is it that the media of mass communication has such a detrimental effect upon us? The problem is the secularism that I mentioned a moment ago.

In 1982 S. Robert Lichter and Stanley Rothman conducted hour-long interviews with 240 journalists and broadcasters at the most influential media outlets including the *New York Times, Washington Post, Time* magazine, *U.S. News and World Report,* CBS, NBC, ABC, PBS, and major broadcasting stations. The results of this survey are startling and confirm even beyond one's worst suspicions, the religious commitment of the media to secularism.[2]

For example, the survey revealed that:

> In a nation in which seven out of ten Americans say they are church members and 60 per cent claim their religious beliefs are very important, 50 per cent of those surveyed had no religions affiliation and 86 per cent seldom or never attended religious services. Little wonder that all things Judaeo-Christian in origin are routinely denigrated. In effect, the media have become the enemy of religious principle and also because of their vast unelected power, the rival of constitutional process and of elected officials.
>
> Granted, if each of us who believe in God is willing to stay in his little corner and do his "religious activities" separated from the arts, industry, alcohol and drug use, education, politics, science, medicine, psychiatry, law, economics, the media, or scholarship — in other words, all that really counts — then he is left to himself. As long as God-fearing people set out to convert souls, fine. But let him stand up and begin to challenge the dominant secularistic forces and the press will make every attempt to either ignore or ravage that individual.
>
> The one thing the media abhors almost without exception is anyone who takes a firm stand on any issue out of religious principle, unless that stand happens to coincide with their expressed views.[2]

Consider for a moment the response of the media when someone dared to question the secularism of the age in which we live.

In 1978 Alexander Solzhenitsyn was asked to give the commencement address at Harvard University. He had this to say about the media:

> How many hasty, immature, superficial and misleading judgments are expressed every day, confusing readers, without any verification? The press can both stimulate public opinion and miseducate it. Thus we may see terrorists turned into heroes, stoned "spaced out" rock

stars and athletes idealized. We read about cocaine as the "in" drug — the drug of beautiful people. We see secret matters pertaining to our nation's defense publicly revealed, or we may witness shameless intrusions on the privacy of well-known people under the slogan: "Everyone is entitled to know everything." Well, this is a false slogan, characteristic of our false era: people also have the right not to know, and it is a much more valuable one. People have the right *not* to have their divine souls stuffed with gossip, nonsense, vain talk. A person who works and leads a meaningful life does not need this excessive burdening flow of information. Hastiness and superficiality are the psychic disease of the 20th century, and more than anywhere else this disease is reflected in the press. In-depth analysis of any problem is anathema to the press. It stops at sensational formulas.

Such as it is, however, the press has become the greatest power within the Western countries, more powerful than the legislature, the executive, and the judiciary. One would then like to ask: By what law has it been elected and to whom is it responsible? In the Communist East, a journalist is frankly appointed as a state official. But who has granted Western journalists their power, for how long a time, and with what prerogatives?[4]

Everybody was delighted when he came from Russia and was very critical of Russian society, but when he started criticizing the Western world, people didn't like it very much. His criticisms were very profound. For example, Solzhenitsyn says:

> If one is right from a legal point of view, nothing more is required; nobody may mention that one could still not be entirely right, urge self-restraint or a renunciation of these rights and call for sacrifice and selfless risk; this would simply sound absurd. Voluntary self-restraint is almost unheard of. Everyone operates at the extreme limit of the legal frames. For example, an oil company is blameless when it buys up an invention for a new type of energy in order to prevent its use. A food-product manufacturer is legally blameless when he poisons his product to make it last longer; after all, people are free not to buy it.[4]

Pornography, violence, sex and other hollow self-serving depictions of the "good life" — almost always associated with alcohol and cocaine — are thrust upon us every day at every turn. How long will we tolerate this corruptive, powerful influence of mass media communications? Then Solzhenitsyn comments on an earlier time saying:

> In American democracy at the time of its birth, all individual human rights were granted on the ground that man is God's creature. That is, freedom was given to the individual conditionally, in the assumption of his constant religious responsibility. Such was the heritage of the preceding 1000 years. Two hundred or even 50 years ago, it would have seemed quite impossible, in America, that an individual be granted boundless freedom with no purpose, simply for the satisfaction of his whim. Subsequently, however, all such limitations were eroded everywhere in the West; a total emancipation occurred from the moral heritage of Judeo-Christian centuries with their great reserves of mercy and sacrifice. State systems were becoming ever more materialistic. The West has finally achieved the rights of men, and even to excess, but man's sense of responsibility to God and society has grown dimmer and dimmer. In just a few short decades, the legalistic selfishness of the Western approach to the world has reached its peak and the world has found itself in a harsh spiritual crisis and a political impasse. All the celebrated technological achievements of progress do not redeem the 20th century's moral poverty, which no one could have imagined even as late as the 19th century.[4]

What Solzhenitsyn says is a tremendously profound and quite accurate indictment of our culture and its moral poverty.

How did the media react to Solzhenitsyn's speech? Did they take it as a tonic, a needed correction to onesided thinking? If anyone speaking to spiritual values might have been heard, it would have been Solzhenitsyn. He was the media's darling; a Nobel Prize winner, the defender of free speech, and the most celebrated literary man to emerge from Russia in the second half of the 20th century. Journalists had, until this time, presented him as a living icon of every cherished Western value. However, listen to this. The *New Yorker,* in their August 21, 1978, issue summarized the media's reaction in their "Notes and Comment Section," without any attempt at so-called liberal-minded fairness.

> While Alexander Solzhenitsyn remained in the Soviet Union, it was possible for us in the United States to regard him as a marvelously steadfast believer in political freedom. But now that we have him in our midst, airing his views on American politics and American society, we have had to review our impressions. We find that primarily he is the champion not of freedom but of spiritual well-being. The two causes are by no means the same.

Olga Carlisle, writing in *Newsweek,* July 24, 1978, said that "Solzhenitsyn's convictions are deeply rooted in the Russian spirit which is untempered by the civilizing influences of a democratic tradition." Let's change that last sentence slightly and see how it sounds. What if we said, "Billy Sundance's convictions are deeply rooted in the American Indian spirit, which is untempered by the civilizing influences of a democratic tradition." What self-respecting secularist would say that? No one. You can't call an American Indian a barbarian (thankfully), but you can say anything about a Christian.

Henry Fairlie, in *The New Republic,* July 29, 1978, depicted Solzhenitsyn as a deranged man with a Messiah complex living in a facsimile of a gulag

prison built to his order in Vermont. About his speech, Fairlie said, "There is nothing new in all of this — it is just the traditional voice of Russian authoritarianism and orthodoxy — and there is nothing for the West to learn from it."[2]

It should be apparent from these responses that the media does not suffer moral confrontation lightly — the confrontation needed today is with the mass media's espousal of a despiritualized irreligious secularistic consciousness.

If secularism were right in declaring that man is born to be happy — as our media would have us believe — then he would not be born to die. Since his body is doomed to die his task on earth evidently must be of a more spiritual nature. It cannot be unrestrained enjoyment of everyday life. It cannot be the search for the best ways to obtain material goods and then cheerfully get the most out of them. It has to be the fulfillment of a permanent, earnest duty, so that one's life journey may become an experience of moral growth, so that one may leave life a better human being than one started it.

Even if we are spared destruction by war, our West will have to change if we want to save life from self-destruction. We cannot avoid revising the fundamental definitions of human life and human society. Do you truly believe that man is above everything? Is there nothing above him? Is it right that man's life and society's activities have to be determined by material expansion in the first place? Is it permissible, is it moral to promote such expansion to the detriment of our spiritual integrity?

If the world has not come to its end, it has approached a major turn in history. It will exact from us a spiritual upsurge, we shall have to rise to a new height of vision, to a new level of life where our physical life will not be cursed as in the Middle Ages, but, even more importantly, our spiritual being will not be trampled upon as in our modern era of secularism. This rising to a new height of vision — a changed world view — will be similar to climbing onto the next anthropological stage. No one on earth has any way left but upward.[4]

What has all this to do with attitudes toward alcohol and other drug use in the U.S.?

Simply this: In a society in which materialism is worshipped, indulgence is encouraged, in which lies and deceit are the accepted norm, in which everything is relative, implying there are no eternal, unchanging values, in which man is the ultimate being, in which speaking to spiritual values in all of life is denigrated, in which there is no demonstration of prudence, justice, fortitude, and courage in our day-to-day lives, in which there is only lip service to our creator, God; in such a society — and we are living in such an age — life has no meaning, no purpose, no direction, and no value.

Is it any wonder that in such a climate people choose to escape the hopelessness and futility of such a world view? Does such a world view explain, at least in part, our current drug crisis? Perhaps we will dare to begin talking with each other about our concerns today and tomorrow that all may leave with something of value, with renewed courage, with hope for the future, and with the conviction that each of us can make a difference in shaping this world of ours to become a more moral, merciful, just, sane, loving, and compassionate place in which to live — and in which our children must live.

Let us begin then today to renew our spiritual values and to work together to think of practical ways, in the area of alcohol and other drug use, to staunch the flow of 20th century's self-righteous inhumanity.

One of our founding fathers said it well: "All that is necessary for the triumph of evil is that good men (and women) remain silent and do nothing."

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Euthanasia

The Role of the Physician

Howard N. Ward, M.D., J.D., *Topeka*

I WAS ONCE advised not to finish law school because given those people who dislike doctors, those who don't like lawyers, and those who can't stand short people, I might not have any friends left. Some people have accused me of going off the deep end by degrees.

My pastor asked if he should be anxious about his position in theology since that might be next, but I assured him that he is completely safe, as it has become apparent that I have already pursued higher education to the point of diminishing returns.

"Euthanasia," "The Right to Die," and more broadly, "Death and Dying," have been of intense interest during the past decade. Perhaps this is because each of us faces death, and in a sense that makes it fascinating as well as distressing. It is the true lowest common denominator, shared with all living creatures. However, even Patrick Henry regarded death as his second choice — "Give me liberty, or give me death." Of course, a person's right to die is not really a legal or medical phenomenon, but a natural phenomenon. In fact, in a primitive society where modern medicine does not exist, a person's right to die goes unchallenged. It is the flowering of modern medicine with its alleged de-emphasis of care and concern, along with medicine's ability to postpone the moment of death and institutionalization of most dying patients, that has perhaps led to such intense interest in these subjects. Some writers seem actually to subscribe to the tenet that the purpose of the medical profession is to prevent a natural death at all costs. Although the average life span has increased from 47 to 73 years in our century, the maximum life span has changed only slightly, so the success of medicine has been primarily in the prevention of premature death rather than extension of the absolute or maximum life span of humans. It is the recognition of that fact that makes it apparent to those involved in the care of dying patients that intensive application of high level and expensive medical technology at the end of a natural life span can, at times, approach absurdity. Also, deplorable conditions sometimes exist in nursing homes and unfortunately, a book entitled *Too Old, Too Sick, Too Bad,* was almost all too correct. This is perhaps another reason why we have become intensely interested in care during the final stages of life. I once heard someone cynically define a nursing home as a finishing school for older girls.

A natural death has something to do with the right to choose one's time and manner of death. Death is a process, not an event, and is an appropriate part of the regenerative cycle. In a minor sense, the person who is not busy being born is busy dying. It is a normal part of the natural order of things, and when understood in that context, it is perhaps not quite so threatening. Someone has said that "Death is nature's way of telling us to slow down."

Although the medical profession has been accused of depersonalizing this experience, there are other entities in our society more deserving of that criticism, the military perhaps being the epitome with death signified by a mere KIA for "killed in action." What permits us, then, to approach a goal of attempting to give life to years rather than mere years to life? The concept of "personhood" is helpful since it deals with an attempt to separate suffering and vegetative life from the pain-free thinking and feeling kind. Qualities that make up personhood include energy, vitality, self-awareness, memory, a sense of futurity, a capacity for interpersonal relationships, love, and desire to live. Near death, one often has only a particle of "personhood."

The word *euthanasia* is derived from two Greek words: *eu,* meaning normal or happy, and *thanatos,* meaning death. Colloquially, it is often used to denote mercy killing. *Dorland's Medical Dictionary* defines *euthanasia* as "an easy or painless death," and the second definition is "mercy killing." Contrariwise, *Black's Law Dictionary,* until the most recent fifth edition, defined it as "the practice of painlessly putting one to death," or the second definition, "an easy or agreeable death." Hence, both professions' dictionaries have a noncriminal meaning of the term, but until recently in reverse order of preference.

Furthermore, euthanasia has been divided into several categories. There can be *active* euthanasia — that is, a deliberate act causing death — or *passive* euthanasia, where one is permitted to die without

This article has been adapted from the text of a speech by Dr. Ward to the General Luncheon on May 7, 1983, at the KMS State Meeting.

medical intervention, due to natural consequences of illness.

There can be sub-categories such as *voluntary,* where it is by the consent of the person involved, or *involuntary,* where it would be without such consent. Thus, if a physician refrained from administering certain medications to a patient at the patient's request, out of respect for a living will that the patient had made, that would be an example of voluntary passive euthanasia.

I believe that active euthanasia — that is, a deliberate act to extinguish life — is outside of the scope of the healing profession, even if it were lawful or ethical. The physician's role and duty in society is to console, alleviate pain and suffering, heal, and to preserve meaningful life. To terminate life directly and immediately would be contrary to that duty and goal. Therefore, if that is ever done, it would be the physician's role to certify that the condition was hopeless, and — if some impartial body agreed with the patient's wishes — some entity other than the physician could extinguish the last remnant of life. If the physician were to serve as both preserver and terminator of life, it could seriously erode the physician-patient relationship. If a physician, in the role of both preserver and terminator, approached the bedside of an extremely ill patient with a syringe full of medication, it could be an anxious experience for all concerned. By analogy, in the legal system, although judge or jury may render a guilty verdict, such trier of fact is not asked to execute the sentence. For similar reasons, physicians should not be participants in capital punishment.

The matter of consent to either passive or active euthanasia can sometimes be more apparent, and perhaps more erroneous, than one might expect. Sometimes depression, an unjustified fear of a specific treatment, an underlying unrecognized problem, and other factors may cloud what appears to be a true consent. For example, an 80-year-old man in a nursing home asked his physician to pray for his death. When the physician returned two weeks later, the old man seemed more anxious than usual, and was quite interested in knowing whether or not the doctor had prayed for his death. When the doctor said "No," the man looked greatly relieved and said, "Shortly after you were here, a very energetic and attractive 80-year-old woman moved in across the hall, and I have completely changed my mind."

Another possible issue is the distinction between merely withholding treatment — that is, never starting it — *vs* withdrawing treatment that has already been started. With respect to withdrawal, there are those treatments that perhaps have become routine such as insulin for a diabetic *vs* those that may have been only recently initiated for a specific illness. Then there can be aggressive and extraordinary therapies such as placing the patient on a mechanical respirator with controlled ventilation. Thus, it becomes rather complicated, but probably measures that are routine and have been administered for years should be continued since the patient expects those to be provided, whereas more recent treatment programs and extraordinary treatments can be withdrawn or withheld in the first place.

How does one derive the authority to exercise this natural right to shape the time and manner of death to the extent possible? Moral theologians say that a person is obligated to accept ordinary treatment, which is defined as treatment that is not burdensome. However, one is not obligated to accept extraordinary treatment, which is defined as unusually burdensome. That is because life itself, although of great value, is not *the* absolute value; if that were true, then death would be *the* absolute evil, and the Judeo-Christian belief does not teach that death is the ultimate or absolute evil. Death, like life, is also a gift from God.

From a legal point of view, there are several bases for this. At common law (case law), Justice Cardozo, in the New York Court of Appeals, stated in a famous case that every human of adult years and sound mind has a right to determine what shall be done with his/her body — that is, a right of self-determination. Thus there is common law that asserts this right.

At the United States Constitutional level, several recent decisions have dealt with the right of privacy. Although this right is not explicitly stated in the Constitution, it is derived from the Bill of Rights. The right of privacy has dealt with such fundamental issues as parenthood and abortion, to mention only two. It appears that the right to die without medical intervention is a part of this right. Within the framework of a free society, one has even the right to be wrong. But legally it is not an absolute right because it must always be balanced against the state's interest in preserving life which includes such issues as safe-guarding health, protecting life, maintaining medical standards, protecting the rights of innocent third parties such as children, and preventing suicide. Hence, there are times when the state's interest would outweigh the right of privacy to determine one's time and manner of death.

An example would be a young mother of four children who is a Jehovah's Witness and who refuses a blood transfusion that could be literally life-

saving. Here, a court might hold that the rights of the innocent children to have a mother outweigh her right to determine her time of death.

Other legal issues that arise include criminal liability, and the physician is sensitive to this in view of his/her role as the most responsible caring figure. At present, active euthanasia would clearly appear to be murder. It makes no difference whether it was for humane purposes or not, since deliberately extinguishing life involves a human-endangering state of mind and thus fulfills the legal definition for malice. With consent, it is still potentially murder, since one cannot consent to be the subject of a criminal homicide. One could even consider possible criminal activity for passive euthanasia if the physician deliberately omitted a medicine, the withdrawal of which hastened the patient's death. This is very theoretical since there are no such cases on record, but it can be argued.

The matter of civil liability appears to be almost entirely theoretical since there are no reported cases dealing with civil liability in euthanasia. But under the wrongful death acts enacted by state legislatures, heirs at law could maintain a cause of action if the deceased could. However, if the heirs requested the form of euthanasia administered, they should not benefit from their own wrongdoing and should not be able to maintain such an action. Therefore, if all family members unanimously agree with the form of care that the patient receives, the physician should usually be free of potential civil liability.

Even defining the moment of death has become difficult. Kansas was the first state to recognize so-called brain death — that is, one can be pronounced legally and medically dead if the traditional findings of death are present: no pulse, no respiration, and no blood pressure, or — in Kansas and about 30 other states — if the patient has no spontaneous brain function. This is of great importance to Kansas physicians because if someone is already legally dead, then one cannot be guilty of any subsequent criminal or civil wrong such as criminal homicide, assisting suicide, or wrongful death.

With respect to determining when someone is dead, Calvin Coolidge was apparently so colorless as a personality, and so diffident and difficult to converse with, that when he died, Dorothy Parker is said to have exclaimed, "How can they tell?"

Perhaps the most erroneous missed diagnosis occurred when Mark Twain was reported to have died while touring in Europe, and he wrote to *The London Daily News,* "The reports of my recent death are greatly exaggerated."

Another legal approach to the right to die has been through legislation. We have previously mentioned the common law right of self-determination espoused by Justice Cardozo and the Constitutional right of privacy. Fifteen states, including Kansas, have now enacted legislation to recognize voluntary passive euthanasia. None of these acts authorize or condone active euthanasia. These acts are based on the cliché, "Death with dignity," if you will.

Not long ago, I read that "It was good that Sam died with dignity. He had lived with so little of it."

The recent legislative acts simply codify the policy that all possible death prevention is not necessarily the wisest course of action. The Kansas legislation, enacted in 1979, authorizes a patient to issue a directive to his/her physician regarding the withholding or withdrawal of life sustaining procedures for certain terminal conditions. In effect, the act authorizes statutorily voluntary passive euthanasia. The physician must comply with the request of the patient as exercised under the act or transfer the patient to a physician who will comply, or be subject to prosecution for unprofessional conduct.

It should be noted that natural death is not necessarily the most comfortable death. The least negative death experience would appear to require adequate symptomatic measures to control pain and suffering.

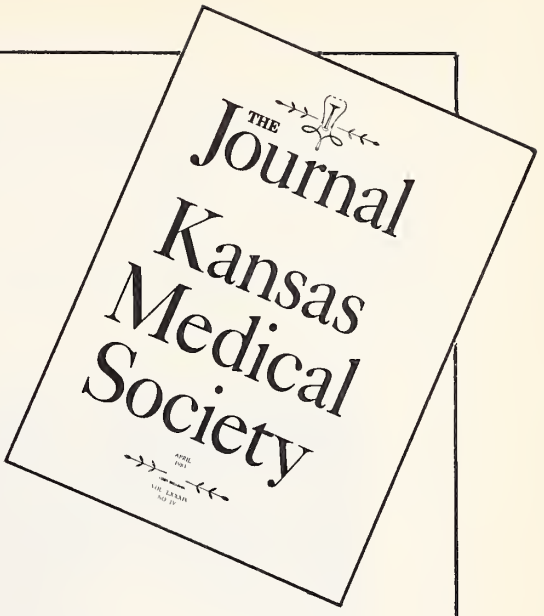
And with respect to choosing a physician, Erma Bombeck's advice is "Beware of the doctor whose office plants die."

During the past six years, several famous cases have received national recognition, with perhaps the most noted being the Quinlan case. Karen Quinlan was a 22-year-old female who had two respiratory arrests of unknown etiology prior to arrival at the hospital, and after aggressive treatment with a respirator for a prolonged period of time, she remained in a persistent vegetative state. The Supreme Court of New Jersey said that she had a right within her constitutional right to privacy to have such extraordinary treatment withdrawn. They furthermore said that since she had become incompetent, such right could be asserted by her guardian, if her guardian, family, physicians, and a hospital ethics committee all concurred in the decision, and there was no reasonable hope or possibility of recovery.

Thus, in New Jersey, if the family, guardian (usually a family member), physicians, and the hospital ethics committee are all in agreement, one can lawfully withdraw or withhold treatment and not be subject to any civil or criminal liability. This was viewed as a friendly case by the medical profession because it left the decision making process where it traditionally has been, although it did add the addi-

(Continued on page 412)

Information for authors . . .



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Council Meeting

Report of Meeting Held April 23, 1983

An extraordinary meeting of the Council was convened by Kermit G. Wedel, M.D., President, for the express purpose of preparing recommendations to the House of Delegates in regard to current changes in health care reimbursement. The meeting was held at the Heart of America Inn, Salina, beginning at 2:00 p.m. Present were Drs.: Carl D. Ambler, Franklin G. Bichlmeier, F. Calvin Bigler, John N. Blank, Kenneth M. Boese, James G. Bridgens, John P. Brockhouse, Clair C. Conard, Louis M. Culp, George Dyck, D. Mikel Elder, Herbert Fransen, Jimmie A. Gleason, Robert J. Haskins, Herman W. Hiesterman, Roman Hiszczynskyj, Lueke B. Lueken, Jack E. Lungstrum, Warren E. Meyer, John R. Neuenschwander, Sr., Lew W. Purinton, Lucien R. Pyle, James H. Ransom, Edwin D. Rathbun, Alex Scott, Newton C. Smith, Thomas F. Taylor, Wayne O. Wallace, Jr., Wallace N. Weber. Also present was Mrs. David G. Laury, President, KMS Auxiliary. Staff present were: Val Braun, Steve Carter, Gary Caruthers.

The Council heard a special report from Pat McGuire, R.N., AMA Liaison with the Federal Government Agencies. She discussed many of the activities that are occurring on the national level, including HMOs, Preferred Provider Organizations and Independent Practice Associations, pointing out the many choices and decisions required of physicians. Ms. McGuire also brought the Council up to date on the issues of AMA-CPT implementation, Professional Review Organizations, mandatory assignment, Diagnostic Related Groupings, ICD-9 and 10, Baby Doe regulations, and the Certificate of Need.

KMS Staff reviewed regulations of the Tax Equity Act, prospective payment mechanisms, and changes in Blue Cross-Blue Shield and Medicaid reimbursements.

Following considerable deliberation, the Council agreed on a proposed KMS Action Plan and prepared a resolution for the House of Delegates. If adopted, the KMS would be directed to educate its members on the changes in health care reimbursement, alternative health care delivery systems, and socio-economic policies of federal and state governmental agencies; reorganize its priorities and focus on legislative concerns; foster closer relationships with hospitals; attract more medical students and residents to KMS membership; cooperate in attempts to cut costs of health care and enhance physicians' image of being the patient's advocate. The resolution requests input and assistance from county medical societies in implementing this action plan.

The meeting adjourned at 5:15 p.m.

Report of Meeting Held May 8, 1983

The Council of the Kansas Medical Society met on Sunday, May 8, 1983, at the Lawrence Holidome immediately following adjournment of the second meeting of the House of Delegates at 10:00 a.m. Jimmie A. Gleason, M.D., President, presided. Those present were Drs.: Carl D. Ambler, Franklin G. Bichlmeier, Kenneth M. Boese, James G. Bridgens, K. William Bruner, Jr., Clair C. Conard, Louis M. Culp, Herbert D. Doubek, Herbert Fransen, Jimmie A. Gleason, Donald D. Goering, David A. Leitch, Wendale E. McAllaster, Warren E. Meyer, John R. Neuenschwander, Sr., Lew W. Purinton, James H. Ransom, Alex Scott, Joan Sehdev, Richard A. Siemens, Newton C. Smith, G. Rex Stone, Max E. Teare, Wayne O. Wallace, Jr., Linda D. Warren, Wallace N. Weber, Kermit G. Wedel, Joel T. Weigand, Emerson D. Yoder. Staff present were Val Braun, Steve Carter, Gary Caruthers.

District #5 invited the Kansas Medical Society to hold its 1987 Annual Meeting in Manhattan.

The Council re-appointed Drs. M. Martin Halley and John A. Segerson to additional three-year terms on the Editorial Board. The Council also formally re-appointed David E. Gray, M.D., as Editor of the *Journal*.

The following new Councilors and Alternates were announced: District #6 — James H. Ransom, M.D., Councilor; Joan Sehdev, M.D., Alternate. District #7 — John P. Brockhouse, M.D., Councilor; W. Scott Ryan, M.D., Alternate. District #14 — Wendale E. McAllaster, M.D., Councilor; Donald L. Wikoff, M.D., Alternate. District #18 — David A. Leitch, M.D., Councilor, Stephen W.

Myrick, M.D., Alternate.

Announcements of all Council meetings will henceforth be sent to Councilors and Alternates.

Edwards, Jackson, and Jefferson counties have reported medical society membership of fewer than four active members. A registered letter will be mailed each individual physician within those counties listing the options provided in the By-Laws for joining an adjacent county medical society. The Council will re-assign membership at its next session. Councilors in the three respective districts were invited to contact members individually.

The meeting adjourned at 10:50 a.m.

Euthanasia

(Continued from page 408)

tional burden of having a hospital ethics committee concur. In that respect, it might be noted that decisions by committees are not always the best decisions.

One might envision a scenario wherein the patient is brought to consciousness momentarily to be advised, "The hospital ethics committee wishes you a speedy recovery by a vote of six to five."

Such committees' consultations should be elective — not mandatory — and their recommendations should be advisory and not binding.

In Massachusetts, a markedly retarded elderly man with acute leukemia became quite ill, and the superintendent of the institution where he had spent his entire life applied for permission to withhold treatment since chances of a response were small, the patient was incapable of being cooperative, and he would have interpreted the pain and discomfort of treatment as perhaps being torturous. As in the Quinlan case, the Court recognized the constitutional right of privacy as permitting a right to die, and prohibiting an unwarranted invasion of bodily integrity. They, likewise, concluded that an incompetent person has that right just as much as a competent person. They also permitted, under the Doctrine of Substituted Judgment, someone else to exercise that right on behalf of the incompetent patient. However, the Court rejected the Quinlan method of reaching the decision of non-treatment. They stated that no group purporting to represent the morality of society, no matter how highly motivated or impressively constituted, could displace the judiciary. The case seemed to say that non-treatment decisions in incompetent patients would have to be adjudicated. This created a raging controversy between medicine and law with physicians accusing judges of ordaining themselves as doctors, and of casting a no-confidence vote against medicine.

A more limited view of the case, and ultimately the correct view, was that the Court would not grant civil and criminal immunity in advance to the figures involved in the care of a patient unless it was adjudicated. If one proceeded with the non-treatment decision without adjudicating it, then that decision would be judged in hindsight. If it were found that such decision was either unlawful or sub-standard, one would be liable for it. However, if the decision was reasonable and lawful, the fact that there was no prior adjudication did not in and of itself create liability. This limited view was expressly stated in a later Massachusetts case. More recently, the Court of Appeals of New York has held that a prior oral statement made by a now incompetent person before such incompetency was a sufficient expression to permit a non-treatment decision.

To displace the traditional decision making triad of patient, physician, and family is to diminish the patient's rights. Brother Fox in New York, and Spring and Saikowicz in Massachusetts, all died prior to conclusion of their legal proceedings. As stated in the Quinlan case, this seems an unnecessary and illogical intrusion upon the field of medicine, which is the arena in which society has traditionally placed this particular responsibility. In essence, where there is a unanimity of opinion between physician and family, and the physician's recommendations conform to acceptable professional standards, this should be the norm on which such decisions are based. It is not necessary to create the situation of medical paternalism *vs* legal imperialism.

Sometimes death comes too soon, but it also sometimes comes too late. And sometimes death is the best that life can offer.

Knee Injuries

(Continued from page 383)

management of these conditions have been discussed. As in all sports injuries, graduated periods of training in running, good footwear and running on satisfactory surfaces, or at least consistently running on the same surface are essential aspects of prevention of knee injuries.

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Drug Resistant Tuberculosis

(Continued from page 391)

Acknowledgement

Fred Tosh, M.D., M.P.H., Director, Sedgwick County Public Health Department, and Gina Barnett, UKSM-Wichita, Coordinator of Curriculum Research, assisted in the preparation of this material.

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Asbestos-Related Disease

(Continued from page 396)

Answers

1. c
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3. d
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KMS Personalities
Annual Meeting, Lawrence, May 5-6







The roving camera picks up highlights of the annual meeting of the KMS at Lawrence as represented by some of the featured dignitaries.

Top: Jimmie Gleason, incoming president, contemplates using his new hammer passed to him by the outgoing president, Kermit Wedel.

Upper left: Dr. Waxman acknowledges the commendation for his service at UKSM, backed by Bill Reals and Kermit Wedel.

Upper right: Joseph Boyle, Chairman of the Trustees of AMA, presents some economic facts of medical life.

Lower left: The Honorable Dan Glickman, Representative, Kansas Fourth District, defends his efforts to curb the PACs.

Lower right: Steve Carter, Executive Director of KMS, proves he has survived his first year of duty.

Below: The annual ritual: President Wedel and Editor David Gray exchange bound volumes of the *Journal*.







The Auxiliary



Meldon Laury retired from her year as esident of KMS Auxiliary with the com- endation of KMS President Kermit edel.



Officers are inducted by Betty Payne, President, AMA Auxiliary. *Left to right:* Diane Sanders, President; Ann Rempel, President Elect; Caryl Bichlmeier, First Vice President; Betty Glover, Second Vice President; Anna Marie McNickle, Recording Secretary; and Phyllis Bigler, Treasurer.



Betty Payne, President of AMA Auxiliary, brings greetings to the Kansas members.

House of Delegates



The meetings of the House of Delegates reflected an intense concern over multiple problems and attracted an overflow crowd of delegates and visitors as the changing medical scene was debated.







Official Proceedings

(Continued from page 366)

M.D., Garnett, and Warren E. Meyer, M.D., Wichita, tellers.

Franklin G. Bichlmeier, M.D., Kansas City, withdrew as a candidate for Vice Speaker because, under the By-Laws, he was ineligible to serve in two positions. Herman Hiesterman, M.D., Chairman of the Nominating Committee, submitted the nomination of Herbert Fransen, M.D., Newton, for the position of Vice Speaker.

The following election results were reported:

PRESIDENT: Jimmie A. Gleason, M.D., Topeka

PRESIDENT ELECT: F. Calvin Bigler, M.D., Garden City

FIRST VICE PRESIDENT: Clair C. Conard, M.D., Dodge City

SECOND VICE PRESIDENT: Franklin G. Bichlmeier, M.D., Kansas City

CONSTITUTIONAL SECRETARY: K. William Bruner, Jr., M.D., Wichita

TREASURER: Wayne O. Wallace, Jr., M.D., Atchison

AMA DELEGATE: Lew W. Purinton, M.D., Wichita

AMA DELEGATE: Kermit G. Wedel, M.D., Minneapolis

AMA ALTERNATE DELEGATE: Jimmie A. Gleason, M.D., Topeka

AMA ALTERNATE DELEGATE: Linda D. Warren, M.D., Hanover

SPEAKER: G. Rex Stone, M.D., Manhattan

VICE SPEAKER: Herbert Fransen, M.D., Newton

The following Council district elections were reported:

DISTRICT 10: Richard Siemens, M.D., Lyons, *Councilor;* Varden Loganbill, M.D., Moundridge, *Alternate*

DISTRICT 12: Carl Ambler, M.D., Pratt, *Councilor;* Joel Weigand, M.D., Wellington, *Alternate*

DISTRICT 16: John Neuenschwander, M.D., Hoxie, *Councilor*

Jimmie A. Gleason, M.D., President, accepted his new responsibilities. The Speaker then announced that the 1984 meeting will be held May 3-6 in Hutchinson.

The meeting was adjourned at 10:00 AM.

HLA Testing from Simple to Complex

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The Midwest Organ Bank Laboratories have changed procedures to provide a less expensive HLA-B27 test. The HLA-B27 test will only check for the presence or absence of the antigen B27. The report will be either B27 positive or B27 negative. This test will include reagents to eliminate confusion with crossreactive antigens that may be mistaken for HLA-B27. The additional antigens tested will include HLA-B7, Bw42, B40, Bw22 and Bw47. All reagents utilized for B27 testing have undergone extensive quality control to ensure potency and accuracy. The charge for this test will be $50.00. The results will be available within 24 hours of the receipt of the blood specimen and, in most cases, on the same day.

Full HLA Profile

The Organ Bank Laboratories will also continue to offer a disease association test. This test should be ordered for any HLA antigen other than HLA-B27. All antigens known to have a disease association are included in the disease association test. The result will provide a full HLA phenotype, listing the antigens found. The cost of this test will be $90.00.

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An asterisk following the resolution number indicates a change in the Constitution and By-Laws.

RESOLUTION NO. 83-1*

Delinquent Dues

Resolved, That the By-Laws be amended by adding new section 11.8111 "A member remaining in arrears as of July 1 of that dues paying year shall be removed from the membership roles."; and be it further

Resolved, That in section 11.83, delete "A member remaining in arrears beyond December 31 of the year in which he became delinquent shall be expelled."; and be it further

Resolved, That in section 11.831, delete "provided that the Executive Committee shall be empowered to consider exceptional cases on an individual basis whether a member shall pay dues for the years in which he/she became delinquent, should he or she desire to renew membership in the future."

RESOLUTION NO. 83-2*

Membership Recruitment Committee

Resolved, That the By-Laws be amended by adding in Section 11.41 between "society" and "any," the words "except as noted in Section 11.411"; and be it further

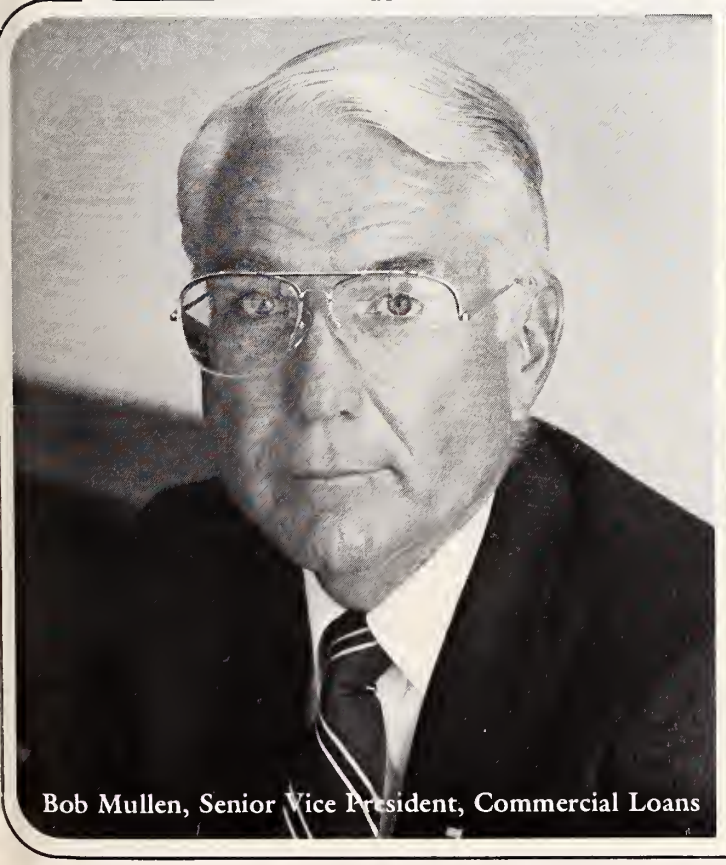
Resolved, That new Section 11.411 be added as follows: "11.411 Medical students and resident physicians shall be eligible for membership in the Kansas Medical Society by applying through the component medical society or by applying directly to the Student or Resident Physician Sections."

RESOLUTION NO. 83-3*

Resident and Medical Student Sections

Resolved, That the By-Laws be amended establishing a Medical Student Section and a Resident Physician Section as sections of the Kansas Medical Society House of Delegates (4.5821); and be it further

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Resolved, That the Medical Student Section and the Resident Physician Section shall be entitled to one delegate and one alternate each to the House of Delegates.

RESOLUTION NO. 83-4

Local Health Departments

WHEREAS, There is an increased need for physician input into identification of appropriate health services to be provided by local health departments; and

WHEREAS, Primary funding of services should be from local resources; and

WHEREAS, Those services that are mandated by the state and conducted by the local health department should be funded by the state; and

WHEREAS, General funding by the state should be provided to finance special problems which transcend county lines and exceed available county resources; therefore be it

Resolved, That a liaison committee be established to consult with the Kansas Department of Health and Environment and the Kansas Local Health Department Association; and be it further

Resolved, That planning and development of public health services be locally developed in accordance with local need; and be it further

Resolved, That physicians representing their local medical societies actively participate in the planning and development of local public health services; and be it further

Resolved, That local medical societies consult with local health departments in the development of protocols specifying what types of services are to be provided and specifying who will provide the services.

RESOLUTIONS NOS. 83-5, 6, 7

Combined with No. 83-4

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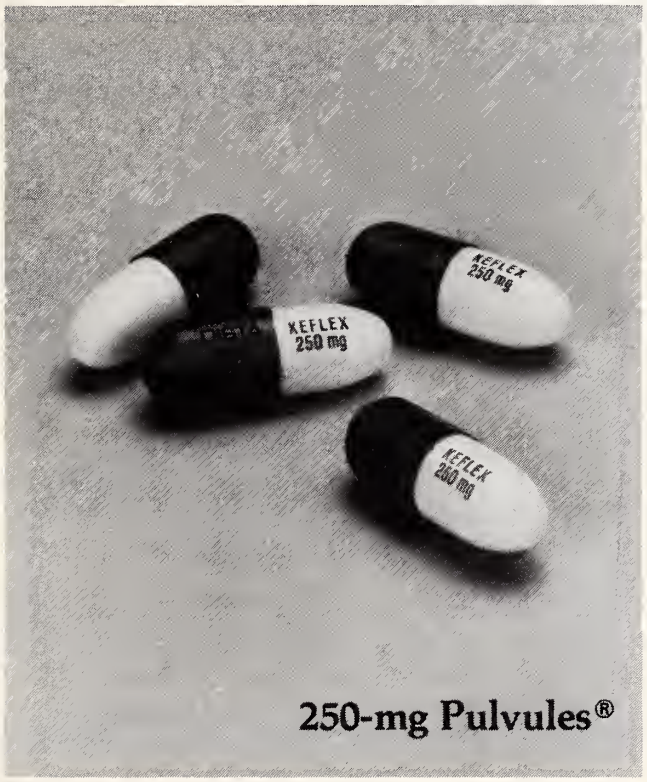
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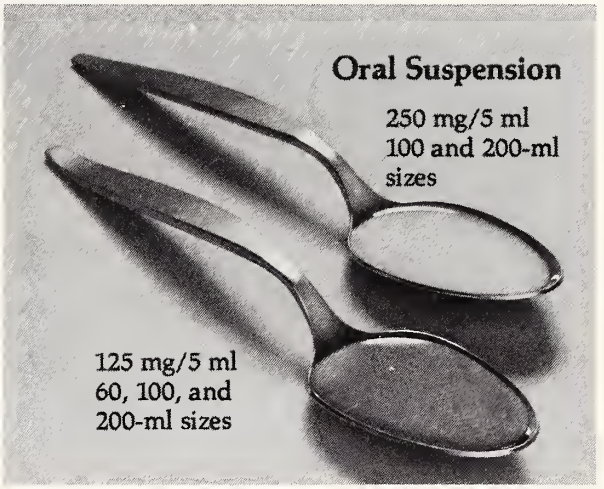
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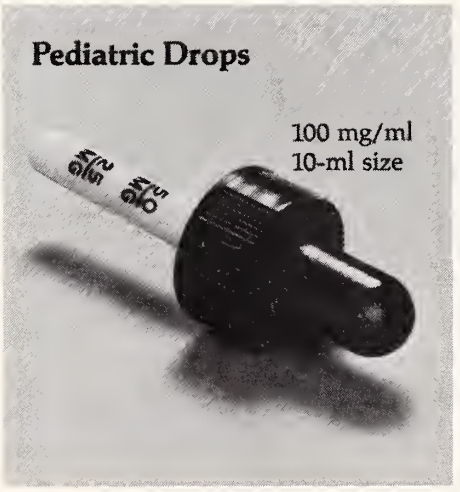
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RESOLUTION NO. 83-8

AMA-CPT

Not adopted.

RESOLUTION NO. 83-9

Hospital Outpatient Clinics

WHEREAS, Hospitals across the country are more and more becoming involved in the private practice of medicine at an accelerating rate; therefore be it

Resolved, That the Kansas Medical Society develop a position paper regarding establishment of hospital outpatient clinics.

RESOLUTION NO. 83-10

Medical Malpractice

Not adopted.

RESOLUTION NO. 83-11

Institutional Reimbursement Policies

Not adopted.

RESOLUTION NO. 83-12

JCAH

WHEREAS, The Joint Commission on Accreditation of Hospitals was formed by the American College of Surgeons, the American College of Physicians, the American Medical Association, the American Hospital Association and the Canadian Medical Association, and has as its mission "to improve the quality of care and services in organized health care settings through a voluntary accreditation process"; and

WHEREAS, At the present time the quality of care and services is assured by numerous factors and agencies; and

WHEREAS, Recent proposals by the JCAH would open the privileges of hospital staff membership to virtually any person claiming to be a provider of health care; and

WHEREAS, Such actions would inevitably lower the standards of medical care which the people of Kansas have come to expect from our hospitals and would certainly increase the overall cost of medical care to the people of Kansas; therefore be it

Resolved, That the Kansas Medical Society declare its opposition to the changes in hospital staff composition that the JCAH and AMA have proposed; and be it further

Resolved, That the Kansas Medical Society encourage its delegates to the AMA to utilize that House of Delegates as a forum to seriously question the desirability of the AMA's continued support of and participation in the JCAH.

RESOLUTION NO. 83-13

Kansas Medical Society Action Plan

WHEREAS, Recent changes in reimbursement for delivery of health care will have a drastic impact on patients, physicians, and hospital providers; and

WHEREAS, Proposed legislative and regulatory changes will implement controlled reimbursement systems and encourage the rapid growth of alternative health care delivery systems; and

WHEREAS, It is critical for physicians to understand these changes and to be informed of future changes in health care financing so as to make appropriate decisions affecting their medical practice; therefore be it

Resolved, That the Council and Executive Committee of the Kansas Medical Society address these issues through the implementation of an action plan which should include but not be limited to:

- Review of current and proposed changes in health care reimbursement, alternative health care delivery systems, and socio-economic policies of federal and state governmental agencies;
- Develop and implement a program to inform Kansas Medical Society members of these policy changes and new systems through the immediate development of a medical-economic manual and the design and support of educational seminars;
- Contract with legal, accounting, and other ancillary services necessary to expedite the implementation of the Kansas Medical Society action plan;
- Select methods of informing patients and the general public of the effects of economic policy changes on the delivery of quality medical care;
- Establish priorities of Kansas Medical Society activities, to include restructuring of committees and staff assignments;
- Reaffirm the Kansas Medical Society commitment to an active liaison with hospitals; and to address issues related to professional liability, Medicare, Medicaid, and allied health professions;
- Request input and assistance from county medical societies to implement this action plan;
- Report action plan progress to the general membership prior to January 1984, and prepare a

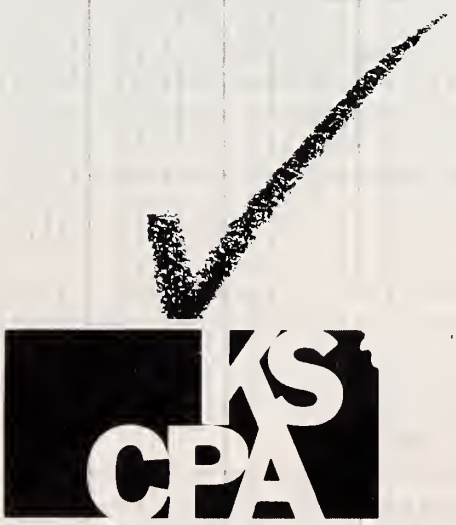


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- ☐ **6** Do you feel it might be time to incorporate your business?
- ☐ **7** Are you preparing a business loan application — and need supporting documentation?
- ☐ **8** Do you want to set up or improve a bookkeeping system?
- ☐ **9** Are you thinking of starting a pension or profit-sharing plan?
- ☐ **10** Do you need an *independent* source of professional management advice on how to use your resources more efficiently or how to make financial projections?

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Kansas Society of Certified Public Accountants

general report reviewing action plan implementation and presenting future policy recommendations to the 1984 meeting of the KMS House of Delegates.

RESOLUTION NO. 83-14

AMA Public Education Program

WHEREAS, Congress has passed or is promoting the Tax Equity and Fiscal Responsibility Act, prospective payment system, and others; therefore be it

Resolved, That the American Medical Association be instructed to immediately launch, *or direct,* a national public educational program informing the citizens of the United States of the consequences of these changes by Congress and the potential threat to the quality of medical care.

RESOLUTION NO. 83-15

Commendation of Dr. Waxman

WHEREAS, David Waxman, M.D., has served the University of Kansas Medical Center as Executive Vice Chancellor since 1977 and as a faculty member since 1961; and

WHEREAS, Dr. Waxman has performed his duties in an exemplary manner and brought great credit to the Medical Center; and

WHEREAS, He has served during a time of great growth both in academic excellence as well as expansion of the Medical Center; and

WHEREAS, Dr. Waxman has supported the Kansas Medical Society in promoting health care for the people of Kansas and supported the private practice of medicine; and

WHEREAS, Dr. David Waxman will retire on July 1, 1983, after a distinguished career; therefore be it

Resolved, That the Kansas Medical Society assembled in the 124th Annual Meeting in Lawrence, Kansas, extend its best wishes and gratitude to Dr. David Waxman for his many contributions to medical education and the State of Kansas; and be it further

Resolved, That copies of this resolution be forwarded to the Governor of Kansas, the Honorable John Carlin; the Chairman of the Kansas Board of Regents; and Chancellor Gene Budig, Kansas University; and be it further

Resolved, That this resolution to commend be spread upon the minutes of the Kansas Medical Society, and that a copy suitably inscribed and adorned be presented to Dr. David Waxman.

RESOLUTION NO. 83-16

KaMPAC

WHEREAS, It is abundantly clear that the political process currently impinges upon the practice of medicine; and

WHEREAS, Among the several valuable avenues for political expression open to Kansas physicians, its political action committee serves a useful purpose; and

WHEREAS, That usefulness is increased in direct proportion to the number of Kansas physicians who participate in KaMPAC; therefore be it

Resolved, That the 1983 Kansas House of Delegates reaffirms its support of KaMPAC and strongly encourages membership therein.

RESOLUTION NO. 83-17

Commendation for the Douglas County Medical Society

WHEREAS, The Douglas County Medical Society and its Auxiliary have done an excellent job in hosting the House of Delegates of the Kansas Medical Society and the Auxiliary to the Kansas Medical Society; and

WHEREAS, They have provided excellent facilities for the meeting and seen to the comfort of the members and guests; and

WHEREAS, The athletic, scientific, and social desires of this Society and its Auxiliary have been fulfilled in grand fashion; therefore be it

Resolved, That the House of Delegates here assembled commend the Douglas County Medical Society and its Auxiliary for their splendid effort by a standing vote of applause; and be it further

Resolved, That copies of this resolution be forwarded to the Douglas County Medical Society and its Auxiliary.

PCP Intoxication

(Continued from page 385)

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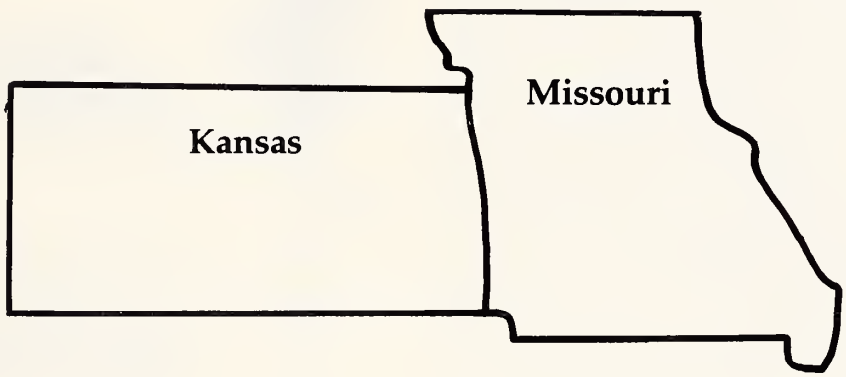
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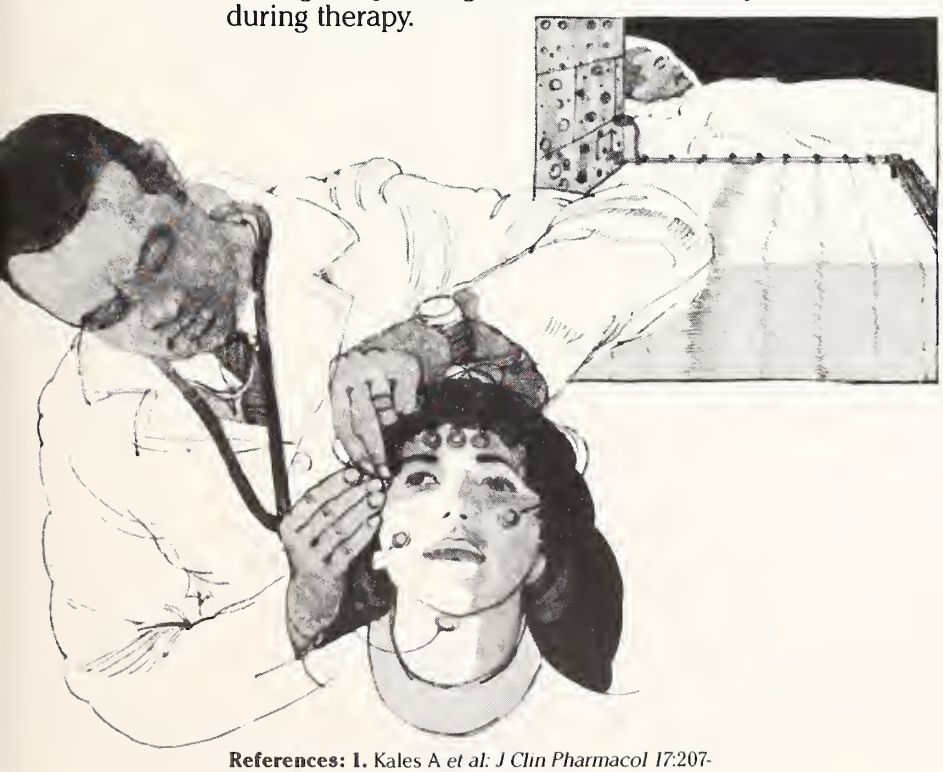
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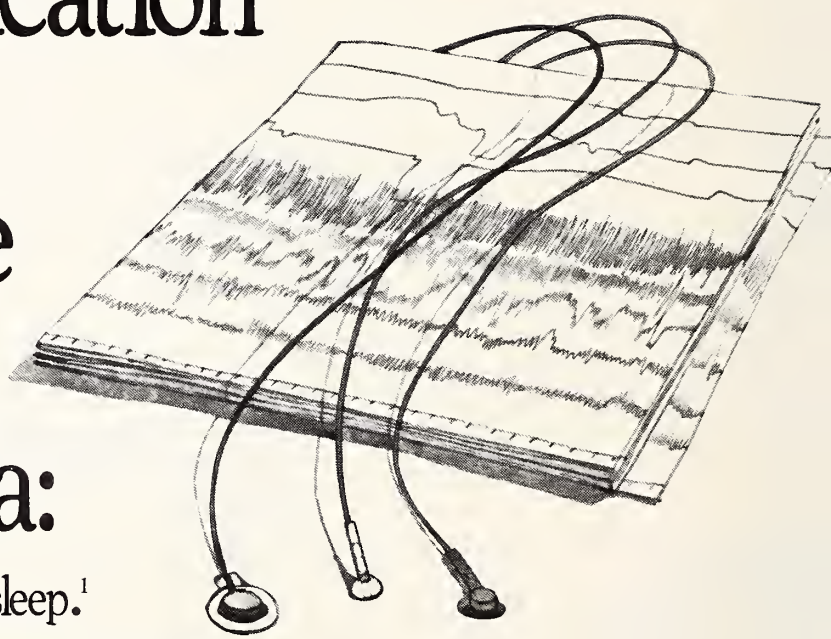
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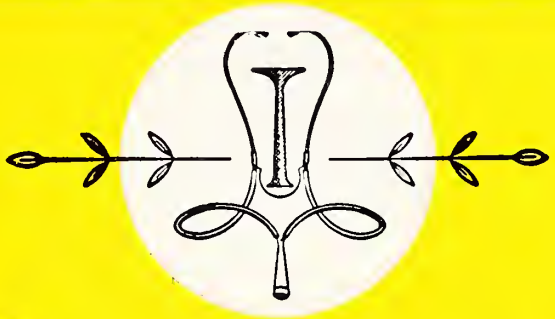
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The JOURNAL of the KANSAS MEDICAL SOCIETY

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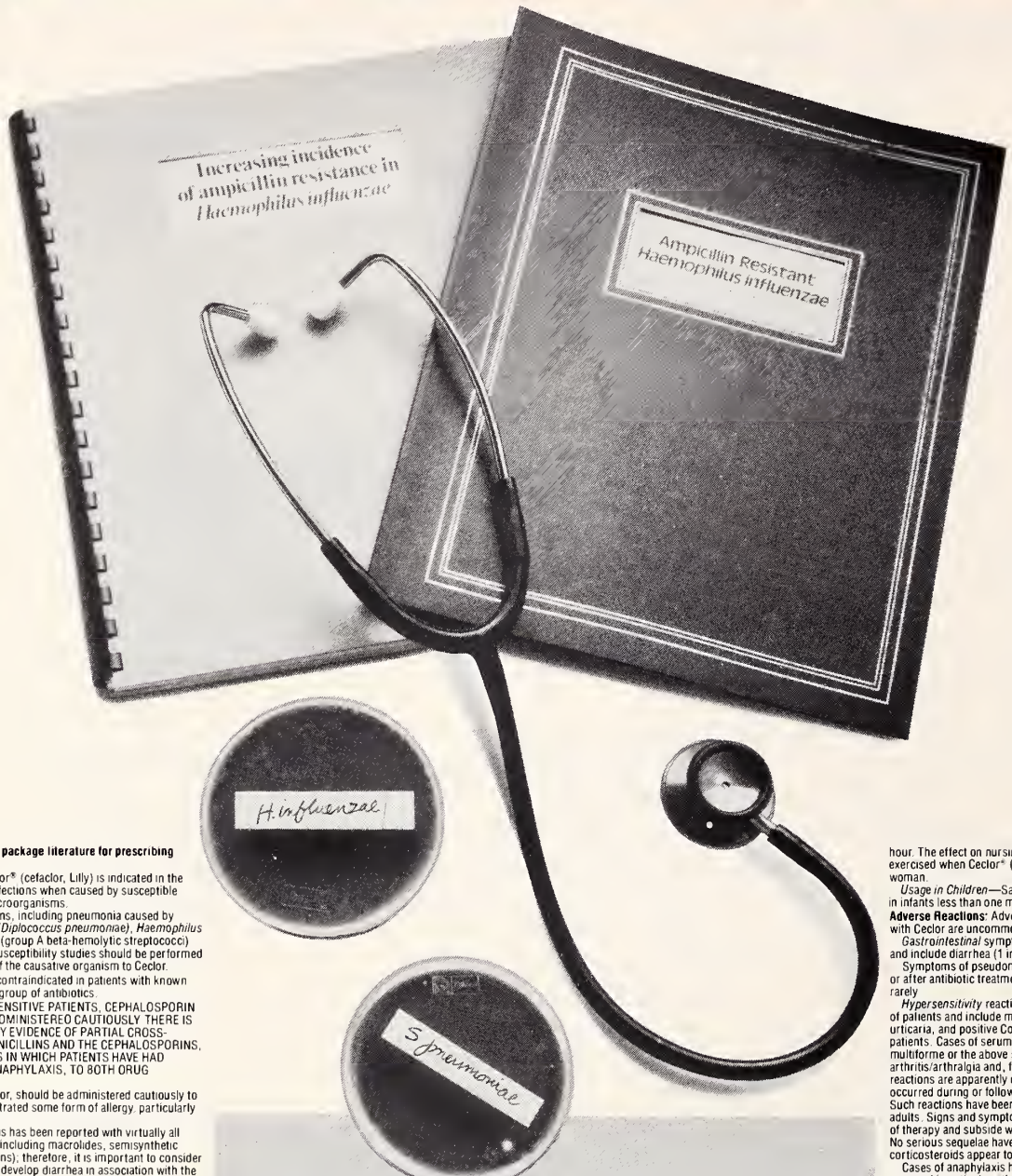
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Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out.

Precautions: *General Precautions*—If an allergic reaction to Ceclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers—Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor® (cefaclor, Lilly) is administered to a nursing woman.

Usage in Children—Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below:

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations of SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

[061782R]

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.*

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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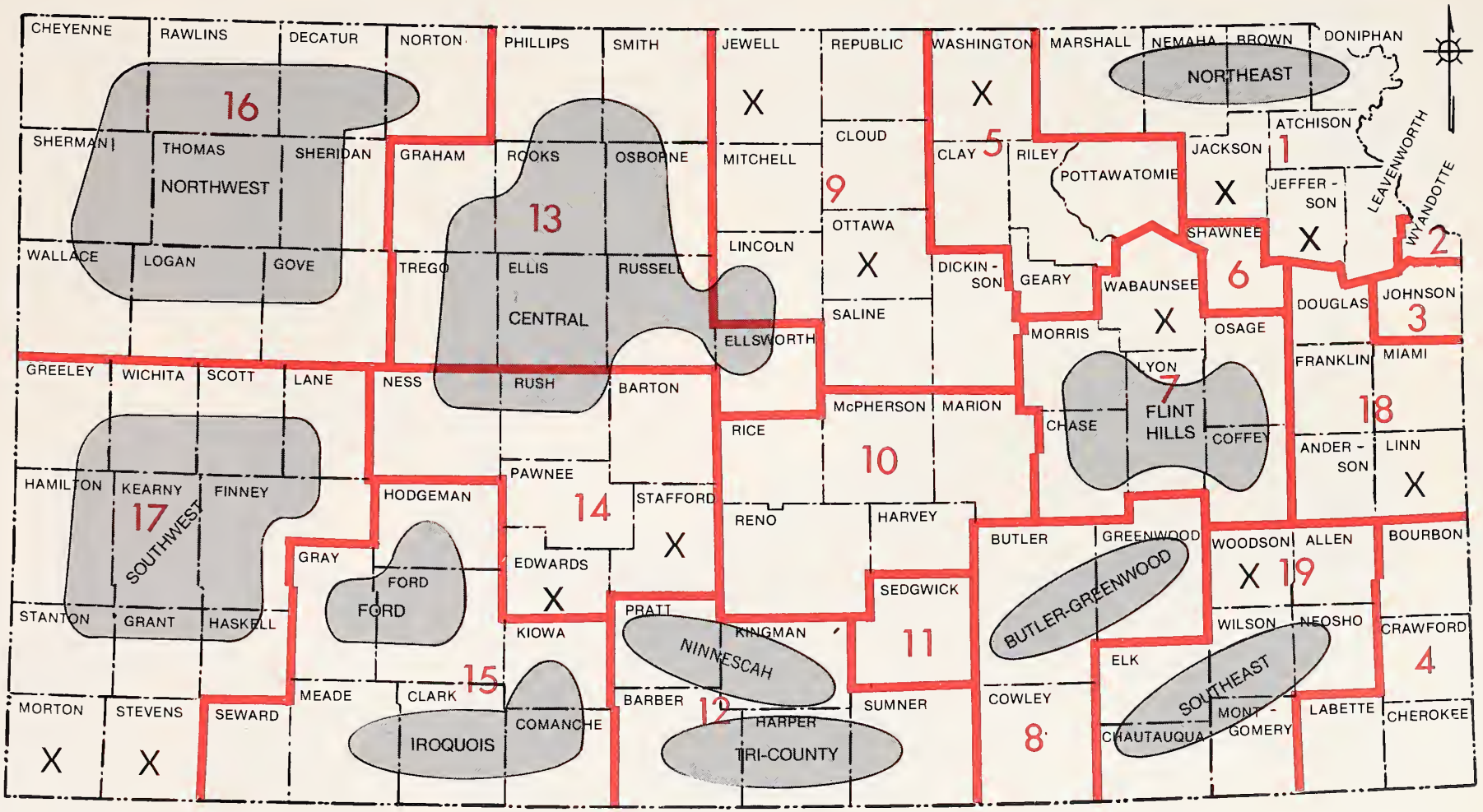
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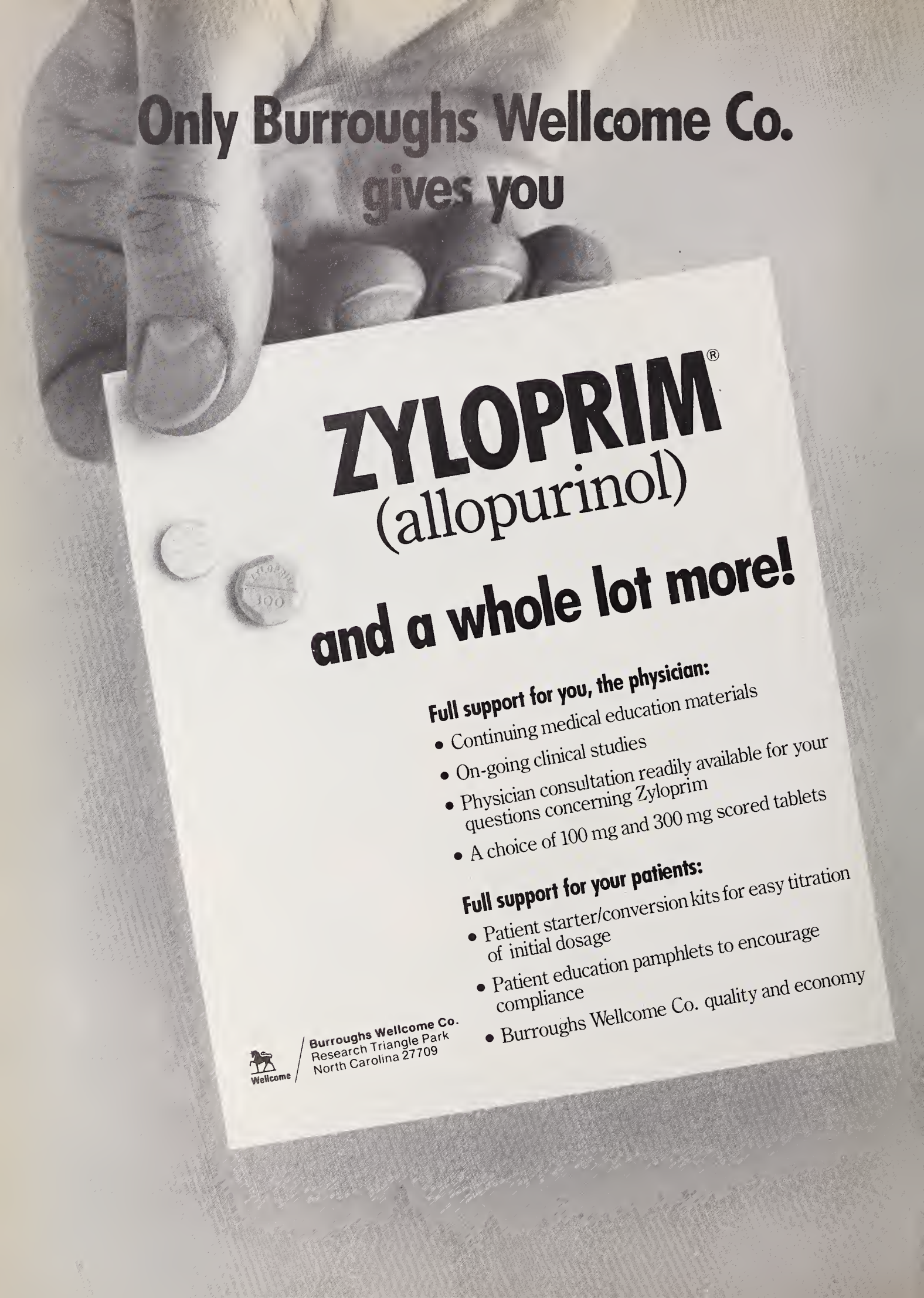
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The Kansas Medical Society has a membership of 2,884 and the Kansas Medical Auxiliary has a membership of 1,274. The following physicians and spouses have elected to become members of KaMPAC and to support "organized medicine" in its efforts to elect "friends of medicine." This listing will be expanded each month to include new KaMPAC members.

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Helpful Hints
on
Audit Procedures

At the expiration date of your policy year, an audit is made by the insurance company to determine the actual payroll amounts, or other exposures during the year. Following this audit, an adjustment may be made which will require additional premium or a return or credit will be ordered. The following are five tips to assist you in preparing for an audit. The following sources will help the auditor:

1. Payroll journal providing monthly totals and division of payroll by type of work performed.
2. Individual earning records indicating the type of work performed. Gross payroll should be totaled by the quarter.
3. Separate record of overtime shown by employee and totaled by class of work for the policy term involved. (Premium for Workers' Compensation is based on straight time pay for all hours worked and does not include ½ extra pay for overtime.) (Not applicable in Delaware, Pennsylvania, and Utah.)
4. Certificates of Workers' Compensation Insurance for all insured sub-contractors.
5. Social Security (Form 941) and State Unemployment Compensation quarterly returns.

Our auditors are instructed to inform you of the date they intend to call on you or to arrange in advance for a convenient time. To assure accurate assignment of your payroll to the proper classes, it is wise for you to arrange to have someone in your organization familiar with employee job assignments available to work with our auditor during the course of the audit.

If your records are kept by an outside accounting firm, make certain the accountants are aware of the impending visit by the auditor so they will have your records available when needed. In the event the accountant is not well informed regarding the duties of various employees, you may wish to brief him in advance of the auditor's visit.

In the audit of your payroll for final billing purposes, you need to determine that the payroll of individual employees is assigned to the appropriate rating classification. This assures that you will be paying the correct premium.

Annual premiums in excess of a specified amount qualify for a discount which varies by state and also by the amount of premium needed to be eligible for the discount. Contact your sales representative if you have any questions about discounts or classifications.

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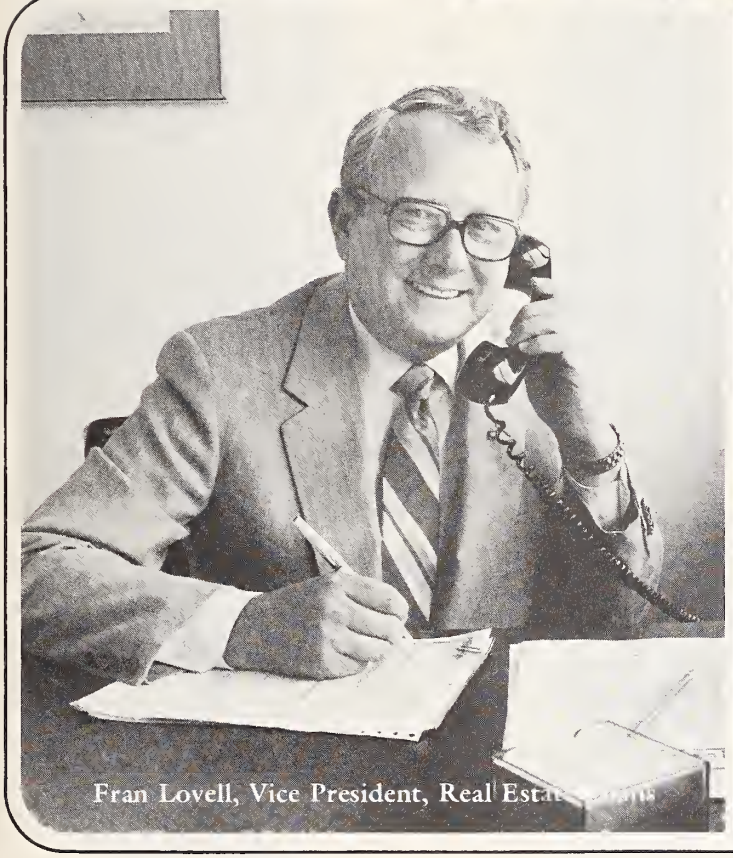
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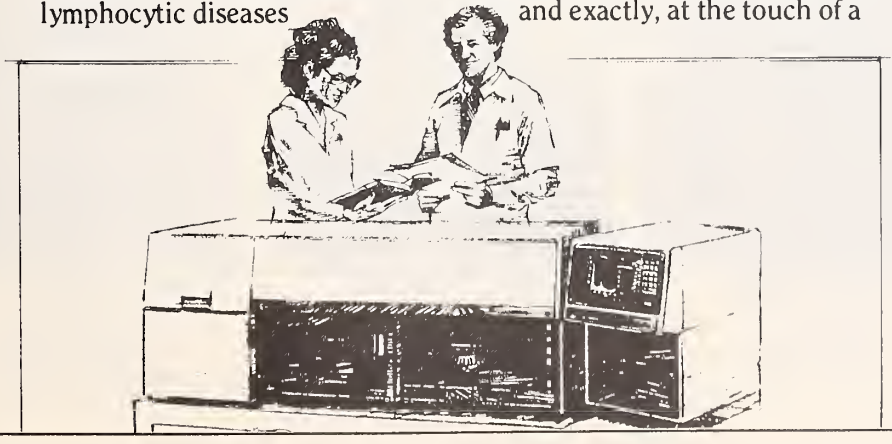
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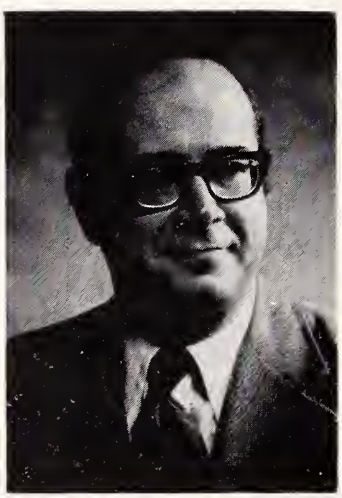
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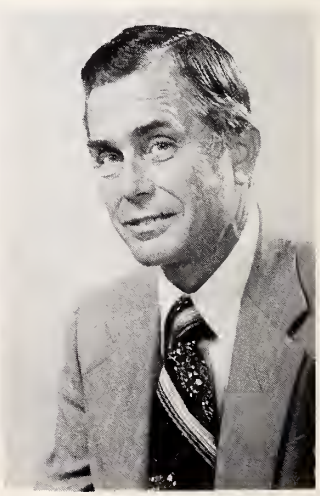
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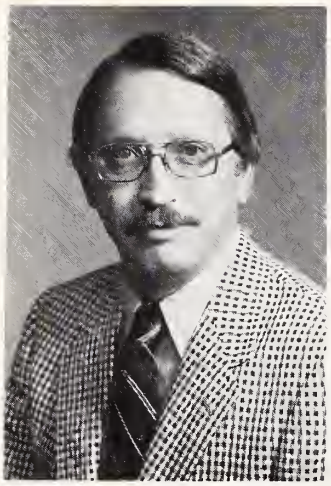
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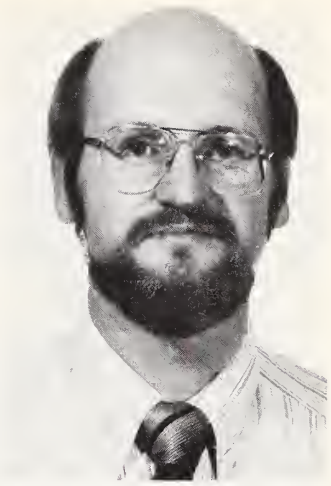
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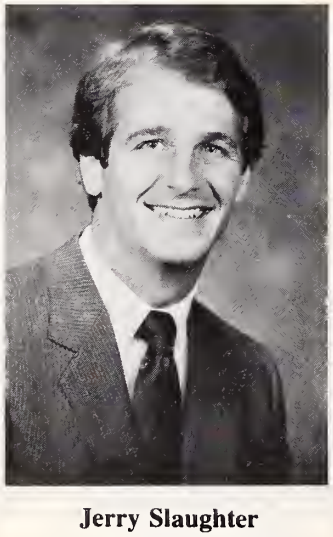


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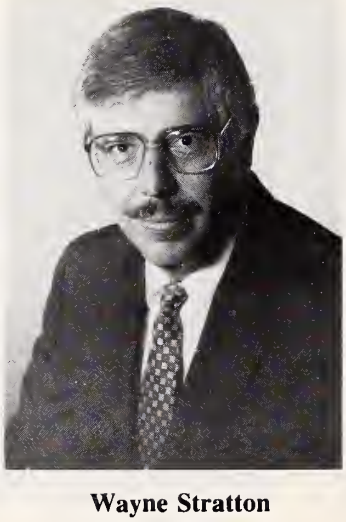


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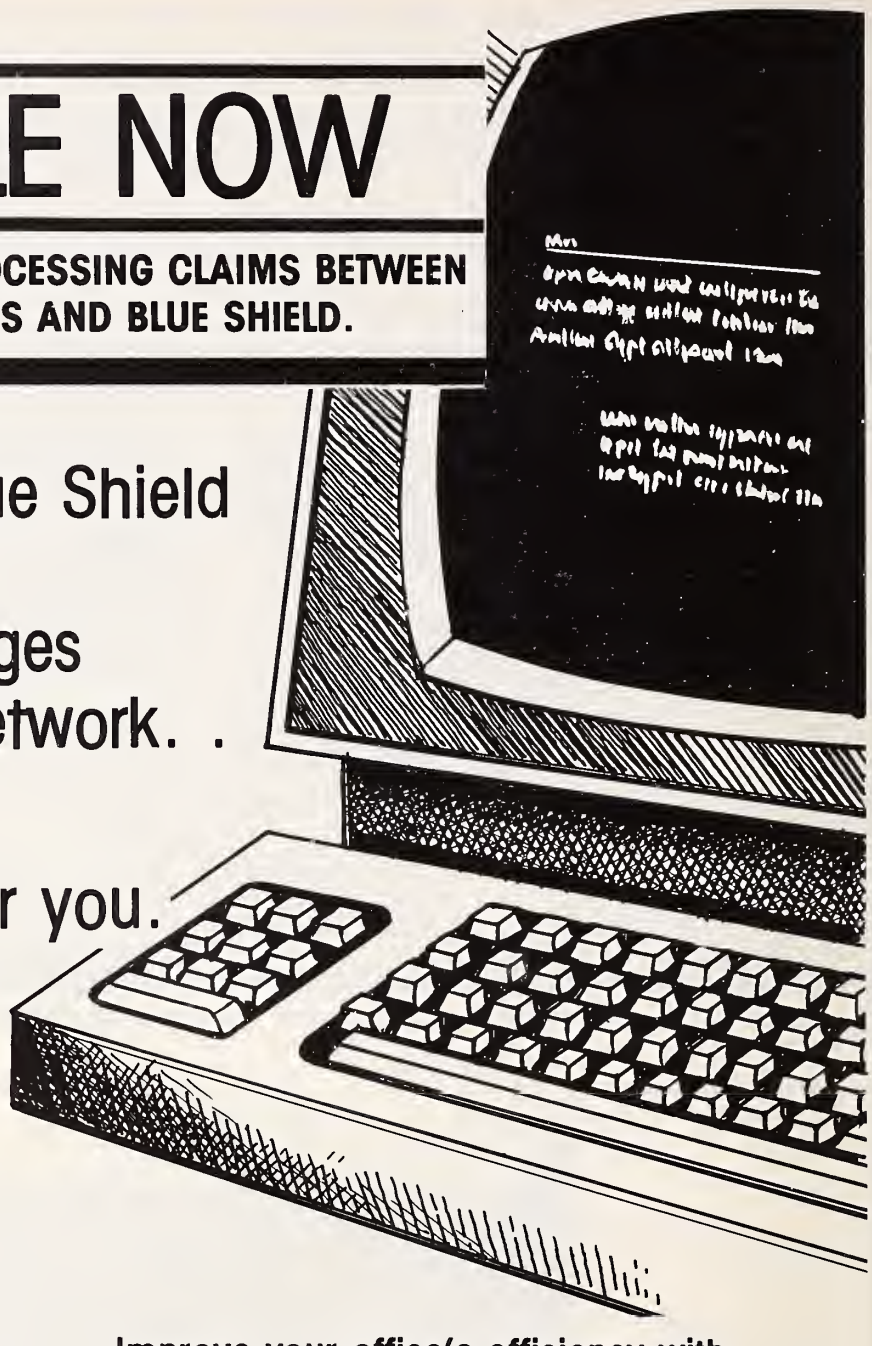
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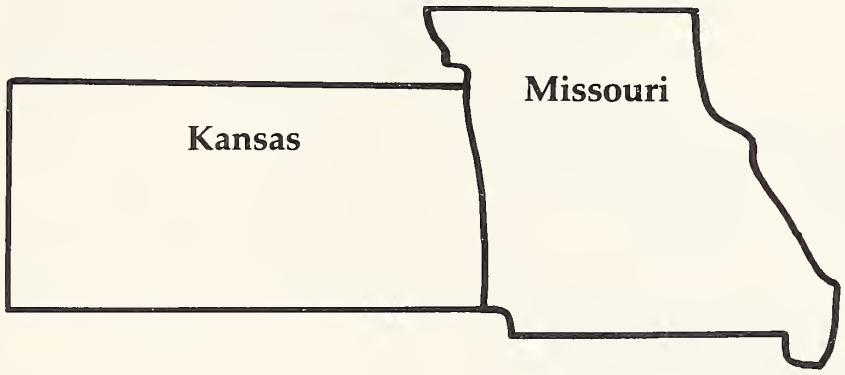
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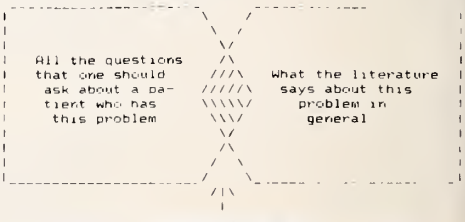
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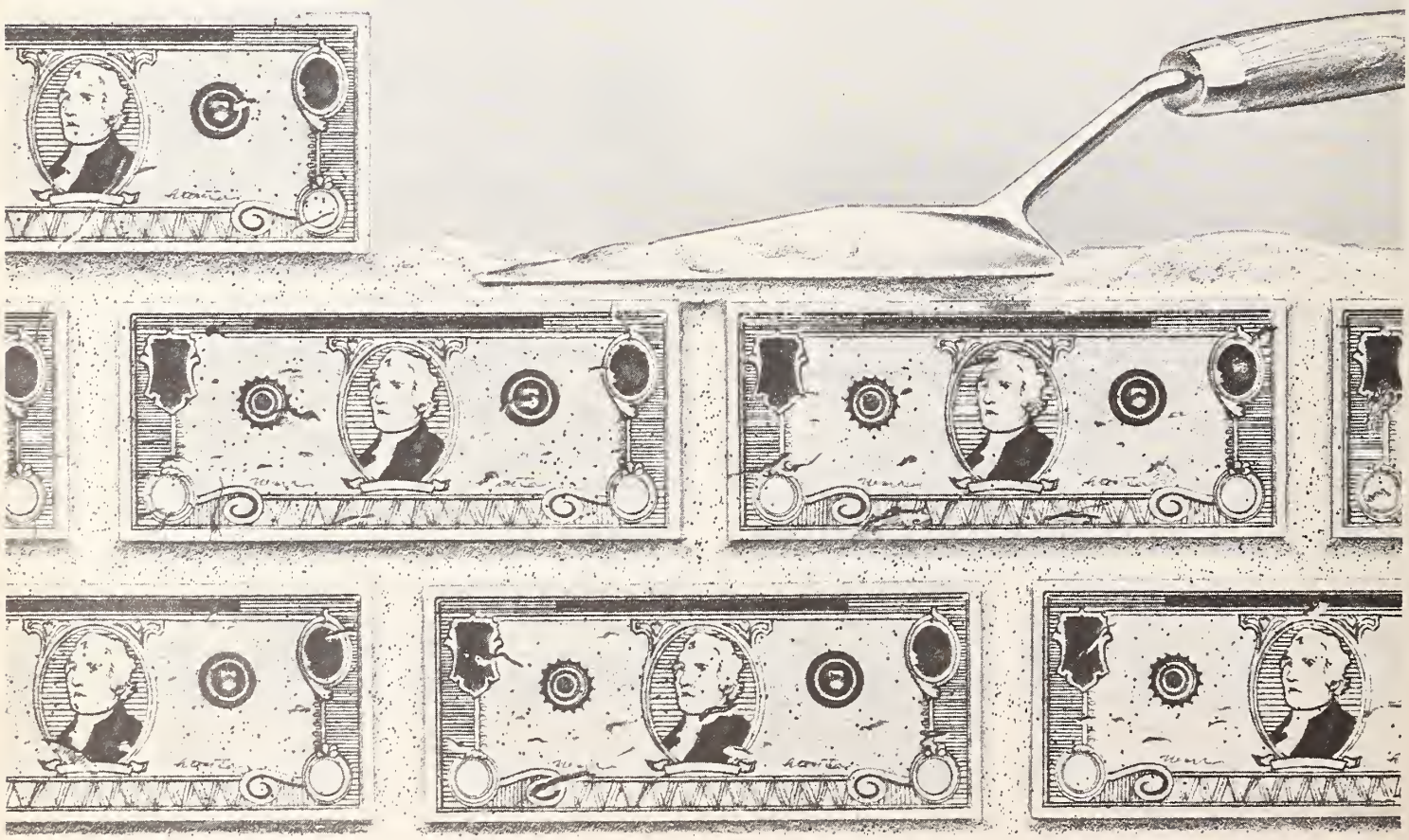
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Hospitals — State Institutions — Poison Control Centers — Home Health Agencies Genetic Counseling Centers

GENERAL HOSPITALS IN KANSAS

Abilene — Memorial, 511 N.E. 10th Street 67410 — 913/262-2100

Anthony — Anthony Hospital & Clinic, 1101 E. Spring 67003 — 316/842-5111

Arkansas City — Arkansas City Memorial, 216 West Birch 67005 — 316/442-2500

Ashland — Ashland District, 709 Oak 67831 — 316/635-2241

Atchison — Atchison, 1301 N. Second 66002 — 913/367-2131

Attica — Attica District, P.O. Box 268 67009 — 316/254-7253

Atwood — Rawlins County, P.O. Box 47 67730 — 913/626-3211

Augusta — Augusta Medical Complex, P.O. Box 430 67010 — 316/775-5421

Baxter Springs — Baxter Memorial, P.O. Box 151 66713 — 316/856-2314

Belleville — Republic County, 66935 — 913/527-2255

Beloit — Mitchell County Community, 400 West 8th 67420 — 913/738-2266

Bucklin — Bucklin District, P.O. Box 38 67834 — 316/826-3211

Burlington — Coffey County, Fourth & Garrettson 66839 — 316/364-2121

Caldwell — Sumner County Dist. #1, 601 So. Osage St. 67022 — 316/845-6492

Caney — Caney Municipal, P.O. Box 325 67333 — 316/879-2182

Cedar Vale — Cedar Vale Retional, P.O. Box 398 67024 — 316/758-2266

Chanute — Neosho Memorial, 629 S. Plummer 66720 — 316/431-4000

Clay Center — Clay County, 617 Liberty 67432 — 913/632-2144

Coffeyville — Coffeyville Memorial, P.O. Box 856 67337 — 316/251-1200

Colby — Citizens Medical Center, 100 E. College Drive 67701 — 913/462-7511

Coldwater — Comanche County, Second & Frisco 67029 — 316/582-2144

Columbus — Maude Norton Memorial City, 220 N. Pennsylvania 66725 — 316/429-2545

Concordia — St. Joseph, 1100 Highland Drive 66901 — 913/243-1234

Council Grove — Morris County, 600 N. Washington 66846 — 316/767-5151

Dighton — Lane County, P.O. Box 728 67839 — 316/397-5321

Dodge City — Dodge City Regional, P.O. Box 1478 67801 — 316/225-9050

El Dorado — Susan B. Allen Memorial, P.O. Box 251 67042 — 316/321-3300

Elkhart — Morton County, P.O. Box 937 67950 — 316/697-2141

Ellinwood — Ellinwood District, 605 North Main 67526 — 316/564-2549

Ellsworth — Ellsworth County Veterans' Memorial, P.O. Box F 67439 — 913/472-3111

Emporia — Newman Memorial County, 12th & Chestnut 66801 — 316/343-6800

Emporia — St. Mary's, Fifteenth & State 66801 — 316/342-2450

Eureka — Greenwood County, 100 East 16th 67045 — 316/583-7451

Fort Scott — Mercy, 821 Burke Street 66701 — 316/223-2200

Fredonia — St. Margaret's Mercy, P.O. Box 519 66736 — 316/378-2121

Garden City — St. Catherine, 608 N. Fifth Street 67846 — 316/275-6111

Gardner — Gardner Community Medical Center, 427 W. Main 66030 — 913/884-8711

Garnett — Anderson County, P.O. Box 309 66032 — 913/448-3131

Girard — Hospital District #1, R.R. #2, Box 5A, 66743 — 316/724-8291

Goodland — Northwest Kansas Medical Center, P.O. Box 629 67735 — 913/899-3625

Great Bend — Central Kansas Medical Center, 3515 Broadway 67530 — 316/792-2511

Greensburg — Kiowa County Memorial, P.O. Box 616 67054 — 316/723-3341

Halstead — Halstead, 328 Poplar 67056 — 316/835-2651

Hanover — Washington County District #1, P.O. Box 38 66945 — 913/337-2214

Harper — Harper County District #5, 12th & Maple 67058 — 316/896-7324

Hays — Hadley Regional Medical Center, 201 East Seventh Street 67601 — 913/628-8251

Hays — St. Anthony, P.O. Box 660 67601 — 913/625-7301

Herington — Herington Municipal, 100 East Helen 67449 — 913/258-2207

Hiawatha — Hiawatha Community, 300 Utah 66434 — 913/742-2131

Hill City — Graham County, P.O. Box 339 67642 — 913/674-2121

Hillsboro — Salem, 701 South Main 67063 — 316/947-3114

Hoisington — Hoisington Lutheran, 250 West Ninth 67544 — 316/653-2114

Holton — Holton City, 510 Kansas Ave. 66436 — 913/364-2116

Horton — Horton Community, P.O. Box 191 66439 — 913/486-2642

Hoxie — Sheridan County, 826 Eighteenth Street 67740 — 913/675-3281

Hugoton — Stevens County, P.O. Box 10 67951 — 316/544-8511

Hutchinson — Hutchinson Hospital Corp., 1701 East 23rd Avenue 67501 — 316/663-6811

Independence — Mercy, P.O. Box 388 67301 — 316/331-2200

Iola — Allen County, 101 South First 66749 — 316/365-3131

Jetmore — Hodgeman County Health Center, P.O. Box 367 67854 — 316/357-8361

Johnson — Stanton County, P.O. Box E 67855 — 316/492-6250

Junction City — Geary Community, P.O. Box 490 66441 — 913/238-4131

Kansas City — Bethany Medical Center, 51 North 12th 66102 — 913/281-8400

Kansas City — Prov.-St. Margaret Health Center, P.O. Box 12430 66112 — 913/596-4000

Kansas City — University of Kansas Medical Center, 39th & Rainbow Blvd. 66103 — 913/588-5000

Kingman — Kingman Community, P.O. Box 376 67068 — 316/532-3147

Kinsley — Edwards County, P.O. Box 99 67547 — 316/659-3621

Kiowa — Kiowa District, 810 Drumm Street 67070 — 316/825-4130

La Crosse — Rush County Memorial, Eighth & Locust 67548 — 913/222-2545

Lakin — Kearny County, P.O. Box 744 67860 — 316/355-7111

Larned — St. Joseph Memorial, 923 Carroll Street 67550 — 316/285-3161

Lawrence — Lawrence Memorial, 325 Maine Street 66044 — 913/749-6100

Lawrence — Watkins Memorial, University of Kansas 66045 — 913/843-4455

Leavenworth — Cushing Memorial, 623 Marshall 66048 — 913/682-8000

Leavenworth — Saint John, 3500 South 4th 66048 — 913/682-3721

Leoti — Wichita County, P.O. Box 968 67861 — 316/375-2233

Liberal — Southwest Medical Center, P.O. Box 1340 67901 — 316/624-1651

Lincoln — Lincoln County, 624 North Second 67455 — 913/524-4403

Lindsborg — Lindsborg Community, 605 West Lincoln 67456 — 913/227-3308

Lyons — Rice County District #1, 619 South Clark 67554 — 316/257-5173

Manhattan — Lafene Student Health Center, Kansas State University 66506 — 913/532-6544

Manhattan — Memorial, P.O. Box 1208 66502 — 913/776-3300

Manhattan — Saint Mary, P.O. Box 1047 66502 — 913/776-3322

Mankato — Jewell County, P.O. Box 327 66956 — 913/378-3137

Marion — St. Luke, 1012 East Melvin 66861 — 316/382-2177

Marysville — Community Memorial, 708 N. 18th Street 66508 — 913/562-2311

McPherson — Memorial, P.O. Box 1166 67460 — 316/241-2250

Meade — Meade District, 510 East Carthage 67864 — 316/873-2141

Medicine Lodge — Medicine Lodge Memorial, 710 North Walnut 67104 — 316/886-3771

Minneapolis — Ottawa County, P.O. Box 209 67467 — 913/392-2122

Minneola — Minneola District, 212 Main 67865 — 316/885-4264

Moundridge — Mercy, 218 East Pack 67107 — 316/345-6391

Neodesha — Wilson County, P.O. Box 360 66757 — 316/325-2611

Ness City — Ness County District #2, 312 Custer 67560 — 913/798-2291

Newton — Axtell Christian, 209 East Broadway 67114 — 316/283-5200

Newton — Bethel Deaconess, P.O. Box 868 67114 — 316/283-2700

Newton — Prairie View Mental Health Center, P.O. Box 467 67114 — 316/283-2400

Norton — Norton County, P.O. Box 250 67654 — 913/877-3351

Norton — Valley Hope Alcoholism Treatment Center, P.O. Box 410 67654 — 913/877-5111

Oakley — Logan County, 211 Cherry Street 67748 — 913/672-3211
Oberlin — Decatur County, 810 West Columbia 67749 — 913/475-2208
Olathe — Mid-Continent Psychiatric, 122 North Cooper 66061 — 913/782-4282
Olathe — Olathe Community, 300 S. Rogers Road 66061 — 913/782-1451
Onaga — Community, 6th & Lucien 66521 — 913/889-4274
Osborne — Osborne County Memorial, 424 West New Hampshire 67473 — 913/346-2121
Oswego — Oswego, Rt. 2, Box 10A 67356 — 316/795-2921
Ottawa — Ransom Memorial, 13th & South Main 66067 — 913/242-3344
Overland Park — Suburban Medical Center, P.O. Box 15959 66215 — 913/492-1000
Paola — Miami County, 501 South Hospital Drive 66071 — 913/294-2327
Parsons — Labette County Medical Center, P.O. Box 767 67357 — 316/421-4880
Parsons — Katy Memorial, 400 Katy 67357 — 316/421-2700
Phillipsburg — Phillips County, P.O. Box 607 67661 — 913/543-5226
Pittsburg — Mt. Carmel Medical Center, Centennial & Rouse 66762 — 316/231-6100
Plainville — Plainville Rural, 304 South Colorado 67663 — 913/434-4553
Pratt — Pratt Regional Medical Center, Third & Commodore 67124 — 316/672-6476
Quinter — Gove County, 5th & Garfield 67752 — 913/754-3341
Ransom — Grisell Memorial Hospital, District #1, P.O. Box 268 67572 — 913/731-2231
Russell — Russell City, 200 South Main 67665 — 913/483-3131
Sabetha — Sabetha Community, 14th & Oregon 66534 — 913/284-2121
St. Francis — Cheyenne County, 210 W. First 67756 — 913/332-2104
St. John — St. John District, 609 East 1st 67576 — 316/549-3255
Salina — Asbury, P.O. Box 1608 67401 — 913/827-4411
Salina — St. John's, P.O. Box 1688 67401 — 913/827-5591
Satanta — Satanta District, P.O. Box 159 67870 — 316/649-2200
Scott City — Scott County, 309 East Third 67871 — 316/872-5811
Sedan — Sedan City, P.O. Box C 67361 — 316/725-3115
Seneca — Nemaha Valley Community, 604 Nemaha 66538 — 913/336-6181
Shawnee Mission — Shawnee Mission Medical Center, 74th & Grandview 66204 — 913/676-2000
Smith Center — Smith County Memorial, 614 South Main Street 66967 — 913/282-6661
Spearville — Spearville District, P.O. Box 205 67876 — 316/385-2661
Stafford — Stafford District, 502 South Buckeye 67578 — 316/234-5221
Syracuse — Hamilton County, P.O. Box 909 67878 — 316/384-7461
Topeka — Memorial, 600 Madison 66607 — 913/354-5100
Topeka — C. F. Menninger Memorial, P.O. Box 829 66601 — 913/273-7500
Topeka — St. Francis Hospital & Medical Center, 1700 West 7th 66606 — 913/295-8000
Topeka — Stormont-Vail Regional Medical Center, 1500 West 10th 66606 — 913/354-6000
Tribune — Greeley County, 308 Greeley 67879 — 316/376-4222
Ulysses — Bob Wilson Memorial, 415 North Main 67880 — 316/356-1266
WaKeeney — Trego County-Lemke Memorial, 320 Thirteenth Street 67672 — 913/743-2134
Wamego — Wamego City, 711 Genn Drive 66547 — 913/456-2295
Washington — Washington County, East Third Street 66968 — 913/325-2211
Wellington — St. Lukes, 1323 North A Street 67152 — 316/326-7451
Wellington — Wellington Hospital & Clinic, 924 South Washington Avenue 67151 — 316/326-3353
Westmoreland — Dechairo Hospital, Inc., First & North Streets 66549 — 913/457-3311
Wichita — Osteopathic, 2622 West Central Avenue 67203 — 316/945-9161
Wichita — St. Francis Regional Medical Center, 929 North St. Francis 67214 — 316/268-5000
Wichita — St. Joseph Medical Center, 3600 East Harry Street 67218 — 316/685-1111
Wichita — Wesley Medical Center, 550 North Hillside 67214 — 316/688-2468
Winchester — Jefferson County Memorial, 408 Delaware St., 66097 — 913/774-4340
Winfield — William Newton Memorial, 1300 East 5th 67156 — 316/221-2300

STATE INSTITUTIONS

Kansas City — Rainbow Mental Health Facility, 2205 W. 36th Street 66103 — 913/384-1880

Larned — Larned State Hospital, P.O. Box 89, Route #3, 67550 — 316/285-2131
Norton — Norton State Hospital, 67654 — 913/877-3301
Osawatomie — Osawatomie State Hospital, P.O. Box 500 66064 — 913/755-3151
Parsons — Parsons State Hospital & Training Center, P.O. Box 738 67357 — 316/421-6550
Topeka — Kansas Neurological Inst., 3107 West 21st Street 66604 — 913/296-5301
Topeka — Topeka State Hospital, 2700 West 6th 66606 — 913/296-4307
Winfield — Winfield State Hospital & Training Center, P.O. Box 548 67156 — 316/221-1200

VETERANS HOSPITALS AND MILITARY HOSPITALS

Ft. Leavenworth — U. S. Munson Army Hospital, Pope and Biddle Avenue 66027 — 913/684-3241
Fort Riley — Irwin Army Comm. Hospital, 66442 — 913/239-3663
Leavenworth — Veterans Administration Medical Center, 66048 — 913/682-2000
Topeka — Veterans Administration Medical Center, 2200 Gage 66622 — 913/272-3111
Wichita — McConnell Air Force Base, USAF Hospital 67221 — 316/685-9254
Wichita — Veterans Administration Center, 5500 E. Kellogg 67218 — 316/685-2221

POISON CONTROL CENTERS

Atchison — Atchison Hospital — 913/367-2131
Dodge City — Trinity Hospital — 316/483-8133
Emporia — Newman Memorial Hospital — 316/342-7120
Fort Scott — Mercy Hospital — Day: 316/223-3100; Night: 316/223-2200
Great Bend — Central Kansas Medical Center — Day: 316/793-3523; Night: 316/792-2511
Hays — Hadley Memorial Hospital — Day: 913/625-2515, Ext. 237; Night: 913/625-3441
Kansas City
University of Kansas Medical Center — 913/588-6633
Bethany Hospital — 913/621-6600
Lawrence — Lawrence Memorial Hospital — Day: 913/843-3680 or 842-4477; Night: 913/843-5874
Parsons — Labette County Medical Center — 316/421-4880
Salina — St. John's Hospital — 913/827-5591, Ext. 222
Topeka — Stormont-Vail Hospital — 913/354-6100
Wichita — Wesley Hospital — 316/688-2222
Kansas Poison Control Information Center: Kansas Department of Health, Food and Drug Division, Topeka — Evan Wright, Director — 913/852-9360, Ext. 541
Antivenin Index Center — 405/271-5454

HOME HEALTH AGENCIES

Anderson County Hospital, Garnett 60032 — 913/448-3131
Barton County Health Dept., Courthouse, Great Bend 67530 — 316/793-7879
Butler-Greenwood County Health Dept., Courthouse, El Dorado 67042 — 316/321-3400
Clay County Hospital, Clay Center 67432 — 913/632-2144
Cloud County Health Dept., Courthouse, Concordia 66901 — 913/243-3588
Coffey County Health Dept., Courthouse, Burlington 66839 — 316/364-5831
Douglas County Visiting Nurses' Association, 342 Missouri St., Lawrence 66044 — 913/843-3738
Ellis County-St. Anthony Hospital, Hays 67601 — 913/625-2556
Ellsworth County Health Dept., Courthouse, Ellsworth 67439 — 913/472-4234
Ford County Health Dept., Courthouse, Dodge City 67801 — 316/225-4991
Franklin County Health Dept., 112 W. Tecumseh, Ottawa 60667 — 913/242-1873
Graham County Hospital, Hill City 67642 — 913/674-2121
Harper County Health Dept., Courthouse, Anthony 67003 — 316/842-5264
Harvey County Health Dept., Courthouse, Newton 67114 — 316/283-1060
Jackson County Health Dept., Courthouse, Holton 66436 — 913/364-2670
Jefferson County Health Dept., Courthouse, Oskaloosa 66066 — 913/863-2447
Kansas City Visiting Nurses' Association, One Gateway Center, Suite 219, Kansas City, Kansas 66117 — 913/371-3770
Kingman County Health Dept., Courthouse, Kingman, 67068 — 316/532-2221
Labette County Health Dept., Box 786, Parsons 67357 — 316/421-4350
Logan County Hospital, Oakley 67748 — 913/672-3211
Lyon County Home Health Service, Newman Memorial Hospital, Emporia 66801 — 316/342-8562

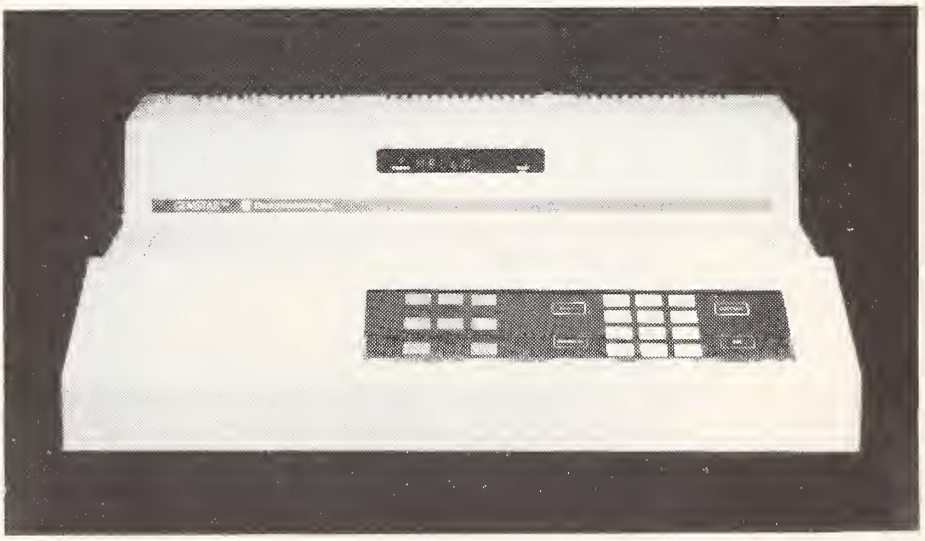
McPherson County Health Dept., Courthouse, McPherson 67460 — 316/241-1753
Morris County Health Dept., Courthouse, Council Grove 66846 — 316/767-5175
Osage County Health Dept., Courthouse, Lyndon 66451 — 913/828-3281
Pratt County Health Dept., 106 E. 2nd, Pratt 67124 — 316/672-6122
Reno County-St. Elizabeth Hospital, 500 W. 20th, Hutchinson 67501 — 316/665-5531
Republic County Health Dept., Courthouse, Belleville 66935 — 913/527-5385
Rice County Health Dept., Courthouse, Lyons 67554 — 316/257-2171
Riley County Health Dept., 616 Poyntz, Manhattan 66502 — 913/776-9721
Russell County Health Dept., Courthouse, Russell 67665 — 913/483-2641
Salina-Saline County Health Dept., 300 W. Ash, Salina 67401 — 913/827-9376
Sedgwick County Health Dept., 1900 E. 9th, Wichita 67214 — 316/268-8433
Shawnee County Health Dept., 1615 W. 8th, Topeka 66606 — 913/233-8961
Sheridan County Hospital, Hoxie 67740 — 913/675-3281
Thomas County-St. Thomas Hospital, Colby 67701 — 913/462-3335
Washington County Health Dept., Washington 66968 — 913/325-2600

GENETIC COUNSELING CENTERS

Kansas City — Genetic Counseling Center, Division of Medical Genetics, UKSM, 39th & Rainbow Blvd., Kansas City, KS 66103 — 913/588-6043
Topeka — Genetic Counseling Center, 1518 SW 8th, Topeka 66606 — 913/232-0957
Wichita — Genetic Counseling Unit, Wesley Medical Center, 550 N. Hillside, Wichita, 67214 — 316/688-2468

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Medical School Codes

UNITED STATES

The following is a list of medical schools in the United States and Puerto Rico, existing and extinct, arranged in state order and showing the code number by which each school is designated in the geographical section of this Directory. Existing approved schools are listed in capital letters.

001 Alabama
001-02 UNIVERSITY OF ALABAMA SCHOOL OF MEDICINE, BIRMINGHAM

003 Arizona
003-01 UNIVERSITY OF ARIZONA COLLEGE OF MEDICINE, TUCSON

004 Arkansas
004-01 UNIVERSITY OF ARKANSAS SCHOOL OF MEDICINE, LITTLE ROCK

005 California
005-02 UNIVERSITY OF CALIFORNIA SCHOOL OF MEDICINE, SAN FRANCISCO
005-06 UNIVERSITY OF SOUTHERN CALIFORNIA SCHOOL OF MEDICINE, LOS ANGELES
005-11 STANFORD UNIVERSITY SCHOOL OF MEDICINE, PALO ALTO
005-12 LOMA LINDA UNIVERSITY SCHOOL OF MEDICINE, LOMA LINDA—LOS ANGELES

007 Colorado
007-02 UNIVERSITY OF COLORADO SCHOOL OF MEDICINE, DENVER

008 Connecticut
008-01 YALE UNIVERSITY SCHOOL OF MEDICINE, NEW HAVEN

010 District of Columbia
010-01 GEORGE WASHINGTON UNIVERSITY SCHOOL OF MEDICINE, WASHINGTON
010-02 GEORGETOWN UNIVERSITY SCHOOL OF MEDICINE, WASHINGTON
010-03 HOWARD UNIVERSITY COLLEGE OF MEDICINE, WASHINGTON

011 Florida
011-02 UNIVERSITY OF MIAMI SCHOOL OF MEDICINE, MIAMI
011-03 UNIVERSITY OF FLORIDA COLLEGE OF MEDICINE, GAINESVILLE

012 Georgia
012-01 MEDICAL COLLEGE OF GEORGIA, AUGUSTA
012-05 EMORY UNIVERSITY SCHOOL OF MEDICINE, ATLANTA

016 Illinois
016-01 RUSH MEDICAL COLLEGE, CHICAGO
016-02 UNIVERSITY OF CHICAGO PRITZKER SCHOOL OF MEDICINE, CHICAGO
016-04 The Hahnemann Medical College and Hospital, Chicago
016-06 NORTHWESTERN UNIVERSITY MEDICAL SCHOOL, CHICAGO
016-11 UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE, CHICAGO
016-42 CHICAGO MEDICAL SCHOOL UNIVERSITY OF HEALTH SCIENCES, CHICAGO
016-43 LOYOLA UNIVERSITY STRITCH SCHOOL OF MEDICINE, MAYWOOD
016-76 CHICAGO COLLEGE OF OSTEOPATHIC MEDICINE, CHICAGO

017 Indiana
017-20 INDIANA UNIVERSITY SCHOOL OF MEDICINE, INDIANAPOLIS

018 Iowa
018-03 UNIVERSITY OF IOWA COLLEGE OF MEDICINE, IOWA CITY
018-75 College of Osteopathic Medicine and Surgery, Des Moines

019 Kansas
019-02 UNIVERSITY OF KANSAS SCHOOL OF MEDICINE, KANSAS CITY

020 Kentucky
020-02 UNIVERSITY OF LOUISVILLE SCHOOL OF MEDICINE, LOUISVILLE
020-12 UNIVERSITY OF KENTUCKY COLLEGE OF MEDICINE, LEXINGTON

021 Louisiana
021-01 TULANE UNIVERSITY SCHOOL OF MEDICINE, NEW ORLEANS
021-05 LOUISIANA STATE UNIVERSITY SCHOOL OF MEDICINE, NEW ORLEANS

022 Maine
022-01 Bowdoin Medical School, Brunswick-Portland

023 Maryland
023-01 UNIVERSITY OF MARYLAND SCHOOL OF MEDICINE, BALTIMORE
023-07 JOHNS HOPKINS UNIVERSITY SCHOOL OF MEDICINE, BALTIMORE

024 Massachusetts
024-01 HARVARD MEDICAL SCHOOL, BOSTON
024-05 BOSTON UNIVERSITY SCHOOL OF MEDICINE, BOSTON
024-07 TUFTS UNIVERSITY SCHOOL OF MEDICINE, BOSTON

025 Michigan
025-01 UNIVERSITY OF MICHIGAN MEDICAL SCHOOL, ANN ARBOR
025-07 WAYNE STATE UNIVERSITY SCHOOL OF MEDICINE, DETROIT
025-12 MICHIGAN STATE UNIVERSITY COLLEGE OF HUMAN MEDICINE, EAST LANSING

026 Minnesota
026-04 UNIVERSITY OF MINNESOTA MEDICAL SCHOOL, MINNEAPOLIS

027 Mississippi
027-01 UNIVERSITY OF MISSISSIPPI SCHOOL OF MEDICINE, JACKSON

028 Missouri
028-02 WASHINGTON UNIVERSITY SCHOOL OF MEDICINE, ST. LOUIS
028-03 UNIVERSITY OF MISSOURI SCHOOL OF MEDICINE, COLUMBIA
028-20 University Medical College of Kansas City
028-22 Ensworth Medical College, St. Joseph
028-34 ST. LOUIS UNIVERSITY SCHOOL OF MEDICINE, ST. LOUIS
028-43 Kansas City College of Medicine and Surgery
028-46 UNIVERSITY OF MISSOURI SCHOOL OF MEDICINE, KANSAS CITY
028-78 Kansas City College of Osteopathy & Surgery
028-79 KIRKSVILLE COLLEGE OF OSTEOPATHIC MEDICINE, KIRKSVILLE

030 Nebraska
030-05 UNIVERSITY OF NEBRASKA COLLEGE OF MEDICINE, OMAHA
030-06 CREIGHTON UNIVERSITY SCHOOL OF MEDICINE, OMAHA
030-07 Nebraska College of Medicine, Lincoln

033 New Jersey
033-05 COLLEGE OF MEDICINE & DENTISTRY OF NEW JERSEY — NEW JERSEY MEDICAL SCHOOL, NEWARK

034 New Mexico
034-01 UNIVERSITY OF NEW MEXICO SCHOOL OF MEDICINE, ALBUQUERQUE

035 New York
035-01 COLUMBIA UNIVERSITY COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK
035-03 ALBANY MEDICAL COLLEGE OF UNION UNIVERSITY, ALBANY
035-06 STATE UNIVERSITY OF NEW YORK AT BUFFALO, SCHOOL OF MEDICINE, BUFFALO
035-08 STATE UNIVERSITY OF NEW YORK COLLEGE OF MEDICINE, BROOKLYN
035-09 NEW YORK MEDICAL COLLEGE, NEW YORK
035-10 Bellevue Hospital Medical College, New York
035-15 STATE UNIVERSITY OF NEW YORK COLLEGE OF MEDICINE, SYRACUSE

035-19 NEW YORK UNIVERSITY SCHOOL OF MEDICINE, NEW YORK
035-20 CORNELL UNIVERSITY MEDICAL COLLEGE, NEW YORK
035-45 UNIVERSITY OF ROCHESTER SCHOOL OF MEDICINE AND DENTISTRY, ROCHESTER
035-46 ALBERT EINSTEIN COLLEGE OF MEDICINE OF YESHIVA UNIVERSITY, NEW YORK

036 North Carolina
036-01 UNIVERSITY OF NORTH CAROLINA SCHOOL OF MEDICINE, CHAPEL HILL
036-05 BOWMAN GRAY SCHOOL OF MEDICINE, WINSTON-SALEM
036-07 DUKE UNIVERSITY SCHOOL OF MEDICINE, DURHAM

038 Ohio
038-02 Eclectic Medical College, Cincinnati
038-06 CASE WESTERN RESERVE UNIVERSITY SCHOOL OF MEDICINE, CLEVELAND
038-40 OHIO STATE UNIVERSITY COLLEGE OF MEDICINE, COLUMBUS
038-41 UNIVERSITY OF CINCINNATI COLLEGE OF MEDICINE, CINCINNATI
038-43 MEDICAL COLLEGE OF OHIO AT TOLEDO, TOLEDO

039 Oklahoma
039-01 UNIVERSITY OF OKLAHOMA SCHOOL OF MEDICINE, OKLAHOMA CITY
039-79 OKLAHOMA COLLEGE OF OSTEOPATHIC MEDICINE AND SURGERY, TULSA

040 Oregon
040-02 UNIVERSITY OF OREGON MEDICAL SCHOOL, PORTLAND

041 Pennsylvania
041-01 UNIVERSITY OF PENNSYLVANIA SCHOOL OF MEDICINE, PHILADELPHIA
041-02 JEFFERSION MEDICAL COLLEGE OF THOMAS JEFFERSON UNIVERSITY, PHILADELPHIA
041-07 MEDICAL COLLEGE OF PENNSYLVANIA, PHILADELPHIA
041-09 HAHNEMANN MEDICAL COLLEGE AND HOSPITAL, PHILADELPHIA
041-12 UNIVERSITY OF PITTSBURGH SCHOOL OF MEDICINE, PITTSBURGH
041-13 TEMPLE UNIVERSITY SCHOOL OF MEDICINE, PHILADELPHIA
041-14 PENNSYLVANIA STATE UNIVERSITY, MILTON S. HERSHEY MEDICAL CENTER, HERSHEY
041-77 PHILADELPHIA COLLEGE OF OSTEOPATHIC MEDICINE, PHILADELPHIA

042 Puerto Rico
042-01 UNIVERSITY OF PUERTO RICO SCHOOL OF MEDICINE, SAN JUAN

043 Rhode Island
043-01 BROWN UNIVERSITY DIVISION OF BIOLOGICAL AND MEDICAL SCIENCES, PROVIDENCE

045 South Carolina
045-01 MEDICAL UNIVERSITY OF SOUTH CAROLINA COLLEGE OF MEDICINE, CHARLESTON

047 Tennessee
047-05 VANDERBILT UNIVERSITY SCHOOL OF MEDICINE, NASHVILLE
047-06 UNIVERSITY OF TENNESSEE COLLEGE OF MEDICINE, MEMPHIS
047-07 MEHARRY MEDICAL COLLEGE SCHOOL OF MEDICINE, NASHVILLE

048 Texas
048-02 UNIVERSITY OF TEXAS MEDICAL BRANCH, GALVESTON
048-04 BAYLOR COLLEGE OF MEDICINE, HOUSTON
048-12 UNIVERSITY OF TEXAS SOUTHWESTERN MEDICAL SCHOOL, DALLAS
048-13 UNIVERSITY OF TEXAS MEDICAL SCHOOL, SAN ANTONIO
048-14 UNIVERSITY OF TEXAS MEDICAL SCHOOL, HOUSTON

049 Utah
049-01 UNIVERSITY OF UTAH COLLEGE OF MEDICINE, SALT LAKE CITY

050 Vermont
050-02 UNIVERSITY OF VERMONT COLLEGE OF MEDICINE, BURLINGTON

051 Virginia
051-01 UNIVERSITY OF VIRGINIA SCHOOL OF MEDICINE, CHARLOTTESVILLE
051-04 MEDICAL COLLEGE OF VIRGINIA HEALTH SCIENCES DIVISION OF VIRGINIA COMMONWEALTH UNIVERSITY, RICHMOND

054 Washington
054-04 UNIVERSITY OF WASHINGTON SCHOOL OF MEDICINE, SEATTLE

055 West Virginia
055-01 WEST VIRGINIA UNIVERSITY SCHOOL OF MEDICINE, MORGANTOWN

056 Wisconsin
056-05 UNIVERSITY OF WISCONSIN MEDICAL SCHOOL, MADISON
056-06 MEDICAL COLLEGE OF WISCONSIN, MILWAUKEE

FOREIGN MEDICAL SCHOOL CODES

CANADA

060 Alberta
060-01 University of Alberta Faculty of Medicine, Edmonton

061 British Columbia
061-01 University of British Columbia Faculty of Medicine, Vancouver

062 Manitoba
062-01 University of Manitoba Faculty of Medicine, Winnipeg

065 Ontario
065-01 University of Toronto Faculty of Medicine, Toronto
065-05 Queen's University Faculty of Medicine, Kingston

067 Quebec
067-01 McGill University Faculty of Medicine, Montreal

OTHER FOREIGN

118 Afghanistan
118-01 Faculty of Medicine, Kabul University, Kabul

132 Argentina
132-01 Facultad de Ciencias Medicas de la Universidad de Buenos Aires, Buenos Aires
132-02 Facultad de Ciencias Medicas de la Universidad Nacional de Cordoba, Cordoba
132-04 Facultad de Ciencias Medicas, Farmacia y Ramos Menores de la Universidad Nacional del Litoral, Rosario, Santa Fe
132-06 Facultad de Ciencias Medicas de la Universidad Nacional de Cuyo, Mendoza, Mendoza

143 Australia
143-03 Faculty of Medicine University of Sydney, Sydney, New South Wales

154 Austria
154-07 Medizinische Fakultat der Universitat Wien, Wien (407-26 from March 13, 1938 to June, 1945)

165 Belgium
165-01 Faculte de Medecine et de Pharmacie Universite libra de Bruxelles, Bruxelles

176 Bolivia
176-02 Facultad de Ciencias Medicas de la Universidad Mayor Real y Pontificia de San Francisco Xavier de Chuquisaca, Sucre
176-03 Facultad de Medicina de la Universidad Mayor de San Simon, Cochabamba

187 Brazil
187-08 Universidade Federal de Parana, Faculdade de Medicina, Curitiba, Parana

215 Cambodia
215-01 Ecole Royal de Medicine Du Cambode, Phnompenh

231 Chile
231-01 Facultad de Medicina de la Universidad de Chile, Santiago

242 China
242 China (also see 243 Effective January 1, 1977)
242-09 St. John's University (Pennsylvania Medical School, Shanghai, Kiangsu (Extinct)
242-16 National Shanghai Medical College, Shanghai, Kiangsu
242-17 West China Union University College of Medicine and Dentistry, Chengtu, Szechuan
242-22 Aurora University Faculty of Medicine, Shanghai, Kiangsu (Extinct)
242-39 Shansi University Medical College, Taiyuan, Shansi

243 China
243-38 National Honan University Medical College, Kaifeng, Honan (242-38 Prior to 1-1-71)
243-51 National Defense Medical Center, School of Medicine, Shanghai, Kiangsu (242-51 Prior to 1-1-71)

244 Taiwan
244 Taiwan (Formosa) effective 1-1-71
244-02 College of Medicine National Taiwan University, Taipai (385-02 Prior to 1-1-71)
244-04 Taipai Medical College, Taipai (385-04 Prior to 1-1-71)

264 Colombia
264-01 Facultad de Medicina de la Universidad Nacional de Colombia Cuidad Universitaria, Bogota, Cundinamarca
264-02 Faculdad de Medicina de la Universidad de Cartagena, Cartagena, Bolivar
264-04 Facultad de Medicina de la Pontificia Universidad Javeriana, Bogota, Cundinamarca
264-06 Facultad de Medicina de la Universidad de Caldas, Manizales, Caldas
264-07 Facultad de Medicina de la Universidad del Cauca, Popayan, Cauca

275 Cuba
275-01 Facultad de Medicina de la Universidad de la Habana, La Habana

308 Dominican Republic
308-01 Facultad de Medicina de la Universidad de Santo Domingo, Ciudad, Trujillo

319 Ecuador
319-01 Facultad de Ciencias Medicas de la Universidad Central, Quito

330 Egypt (United Arab Republic)
330-02 Kasr-el-Aini-Faculty of Medicine Cairo University, Cairo (Formerly Fouad First University Faculty of Medicine)
330-03 Faculty of Medicine Alexandria University, Alexandria

341 El Salvador
341-04 Facultad de Medicina Universidad Nacional del Salvador, San Salvador

352 England
352-05 School of Medicine University of Leeds, Leeds
352-07 University of London Faculty of Medicine, London
352-11 Registrable Qualifications granted by English Conjoint Board (Royal College of Surgeons of England/Royal College of Physicians of London)

385 Formosa (Taiwan)
385 (Also see 244 Taiwan [Effective 1-1-71])
385-01 Kaohsiung (takau) Medical College, Kaohsiung
385-02 College of Medicine National Taiwan University, Taipai
385-03 National Defense Medical Center, Taipai
385-05 China Medical College, Taichung

396 France
396-06 Faculte de Medecine de l'Universite de Paris, Paris, Seine
396-07 Faculte mixte de Medecine et de Pharmacie de l'Universite de Toulouse, Toulouse, Haute-Garonne

407 Germany
407 Also see 408-409—East and West Germany (Effective 1-1-71)
407-07 Medizinische Fakultat der Georg-August-Universitat, Gottingen, Niedersachsen
407-10 Medizinische Fakultat der Universitat Heidelberg, Heidelberg, Baden-Wurttemberg
407-15 Medizinische Fakultat der Phillipps-Universitat, Marburg/Lahn, Hessen
407-16 Medizinische Fakultat der Ludwig Maximiliams-Universitat, Munchen, Bayern
407-21 Medizinische Fakultat der Universitat Hamburg, Hamburg
407-23 Medizinische Fakultat der Johann-Wolfgang-Goethe-Universitat, Frankfurt-Am-Main, Hessen
407-33 Medizinische Fakultat der Freien Universitat Berlin, Berlin

409 Germany West
409-05 Medizinische Fakultat Albert-Ludwigs-Universitat Freiberg IM Breisgau
409-33 Medizinische Fakultat Freien Universitat, Berlin, Berlin (407-33 Prior to 1-1-71)

418 Greece
418-01 Faculty of Medicine National University of Athens, Athens
418-02 Faculty of Medicine University of Thessaloniki, Thessaloniki

429 Guatemala
429-01 Facultad de Ciencias Medicas, Universidad de San Carlos, Guatemala

451 Honduras
451-01 Facultad de Medicine y Cirugia de la Universidad Nacional Autonoma de Honduras, Tegucigalpa

473 Hungary
473-01 Orvosi Fakultas Tudomanyegyetem, Budapest

495 India (Goa)
495-01 University of Bombay, Affiliated Medical Colleges are:
a. Grant Medical College Bombay University, Bombay, Maharashtra
b. Seth Gorhandas Sunderdas Medical College Bombay University, Bombay, Maharashtra
495-04 Madras Medical College Madras University, Madras, Madras
495-08 Christian Medical College Punjab University, Ludhiana, Punjab
495-09 St. John's Medical College, Bangalore, Mysore (before June 1966: Government Medical College, Mysore University, Mysore)
495-11 Andhra Medical College Andhra University, Visakhapatnam, Andhra
495-16 Stanley Medical College Madras University, Madras, Madras
495-18 Assam Medical College Gauhati University, Dibrugarh, Assam
495-21 Osmania Medical College Osmania University, Hyderabad, Andhra
495-23 Medical College Baroda University, Baroda, Gujarat
495-27 Christian Medical College, Vellore, Madras
495-28 Byramjee Jeejeebhoy Medical College, Poona, Maharashtra
495-29 Government Medical College Punjab University, Patiala, Punjab
495-30 Sawai Man Singh Medical College Rajasthan University, Jaipur, Rajasthan
495-31 Medical College Kerala University, Trivandrum, Kerala
495-34 Gajra Rajo Medical College Vikram University, Gwalior, Madhya Pradesh
495-35 Karnatak Medical College Karnatak University, Hubli, Mysore
495-36 All-India Institute of Medical Sciences, New Delhi, Delhi
495-37 Kasturba Medical College Karnatak University, Manipal, Mysore
495-41 G.S.V. Memorial Medical College Lucknow University, Kampur, Uttar Pradesh
495-47 Medical College Jabalpur University, Jabalpur, Madhya Pradesh
495-48 M.P. Shah Medical College Gujarat University, Jamnagar, Gujarat
495-49 Ghandhi Medical College Vikram University, Bhopai, Madhya Pradesh
495-50 Guntur Medical College Andhra University, Guntur, Andhra

495-54 Rajendra Medical College, Ranchi, Bihar
495-55 Sardar Patel Medical College, Bikaner, India
495-57 Kakatiya Medical College, Warangal, Andhra Pradesh
495-96 Lokmanya Tilak Mun Medical College, Bombay University, Bombay, Maharashtra
495-97 Dr. Vaishampayan Memorial Medical College, Shivaji University, Shalopur, Maharashitra

496 India
496-11 Sri Krishna Medical College, Muzaffarpur, Bihar

506 Indonesia
506-02 Faculty of Medicine Airlangga Airlangga University, Surabaya

517 Iran
507-01 Faculty of Medicine University of Teheran, Teheran

528 Iraq
528-01 Faculty of Medicine Baghdad University, Baghdad

539 Ireland
539-01 Faculty of Medicine Queen's University of Belfast, Belfast
539-02 National University of Ireland, Constitient Colleges are:
a. Faculty of Medicine University College, Dublin
b. Faculty of Medicine University College, Cork
c. Faculty of Medicine, Galway

550 Israel
550-01 The Hebrew University-Hadassah Medical School, Jerusalem

561 Italy
561-15 Facolta di Medicina e Chirurgia dell'Universita di Perugia, Perugia
561-19 Facolta di Medicina e Chirurgia dell'Universita di Siena, Siena

572 Japan
572-11 Tokyo Medical College (Nippon Ikadaigaku) Hongo, Tokyo (Extinct)
572-41 Faculty of Medicine Shinshu University, Matsumoto, Nagano
572-49 Tokyo Medical College, Tokyo

583 Korea (South)
583-01 Severence Medical College Yonsei University, Seoul
583-02 College of Medicine Seoul National University, Seoul
583-03 Korea University Medical College, Seoul
583-04 College of Medicine Kyong-Puk National University, Taegu
583-06 College of Medicine Chun Nam National University, Kwangiu
583-09 College of Medicine Pusan National University, Pusan
583-10 College of Medicine Catholic University, Seoul

605 Lebanon
605-01 Medical School American University of Beirut, Beirut

627 Malta
627-01 Faculty of Medicine and Surgery Royal University of Malta, Valetta

649 Mexico
649-01 Facultad de Medicina de la Universidad Nacional Autonoma de Mexico, Mexico
649-02 Facultad de Medicina de la Universidad de Nuevo Leon, Monterrey, Nuevo Leon
649-06 Facultad de Medicina de la Universidad Nacional del Sureste, Merida, Yucatan
649-14 Facultad de Medicina de la Universidad Autonoma de Guadalajara, Guadalajara, Jalisco
649-36 Centro de Estudios Universidad Xochicalo A.C., Cuernavaca, Morelos

660 Netherlands
660-61 Faculteit der Geneeskunde Universiteit Van Amsterdam, Amsterdam

671 New Zealand
671-01 Medical School University of Otago, Dunedin

704 Pakistan
704-01 King Edward Medical College, Lahore, West Pakistan
704-02 Dow Medical College, Karechi, Federal Capital Area
704-03 Dacca Medical College, Dacca, East Pakistan
704-04 Nishtar Medical College, Multan, West Pakistan
704-09 Khyber Medical College, Peshawar, North-West Frontier Province
704-10 Chittagong Medical College, Chittagong, East Pakistan (160-01 after 7-1-72)

726 Paraguay
726-01 Facultad de Medicina de la Universidad Nacional de Asuncion, Asuncion

737 Peru
737-01 Facultad de Medicina de San Fernando de la Universidad Nacional Mayor de San Marcos, Lima
737-05 Facultad de Medicina de la Universidad Nacional de San Agustin, Arequipa
737-06 Facultad de Medicina "Cayetano Heredia" de la Universidad Peruana de Ciencias Medicas y Biologicas, Lima

748 Phillipines
748-01 Faculty of Medicine and Surgery University of Santo Tomas, Manila
748-02 College of Medicine University of the Phillipines, Manila
748-07 College of Medicine Manila Central University, Manila
748-08 Institute of Medicine Far Eastern University, Manila
748-09 College of Medicine Southwestern University, Cebu City
748-10 College of Medicine University of the East, Quezon City
748-11 College of Medicine Cebu Institute of Technology, Cebu City

803 Scotland
803-01 Faculty of Medicine University of Aberdeen, Aberdeen
803-03 Faculty of Medicine University of Edinburgh, Edinburgh

836 South Africa
836-01 Medical School University of the Witwatersrand, Johannesburg

847 Spain
847-01 Facultad de Medicina de la Universidad de Barcelona, Barcelona
847-04 Facultad de Medicina de la Universidad de Madrid, Madrid
847-06 Faculdad de Medicina de la Universidad de Zaragoza, Zaragoza
847-08 Facultad de Medicina de la Universidad de Valencia, Valencia
847-10 Facultad de Medicina de la Universidad de Salamanca, Salamanca
847-11 Facultad de Medicine de la Universidad Catolica Navarra, Pamplona

869 Switzerland
869-01 Medizinische Fakultat der Universitat Basel, Basel
869-02 Medizinische Fakultat der Universitat Bern, Bern
869-05 Faculte de Medecine de l'Universite de Lausanne, Lausanne

875 Syria
875-01 Faculty of Medicine Damascus University, Damascus

Taiwan (See Formosa)

891 Thailand
891-01 Faculty of Medicine at Chulalongkorn Hospital University of Medical Sciences, Bangkok
891-02 Faculty of Medicine at Sariraj Hospital University of Medical Sciences, Thonburi
891-04 Faculty of Medicine at Ramathibodi Hospital, Mahidol University, Bangkok

902 Turkey
902-01 Tip Fakultesi Istanbul Universitesi, Istanbul
902-05 Haceteppe University Faculty of Medicine, Ankara

913 Union of Soviet Socialist Republics
913-02 Voronez Medical Institute, Voronez

941 Viet-Nam South
941-01 Faculte mixte de Medicine et de Pharmacie Universite de Saigon, Saigon

957 Yugoslavia
957-02 Medicinski Fakultet Univerziteta u Beogradu, Beograd

Alphabetical Listing

A

ABBAS, DILAWER H, WICHITA
ABBUEHL, DON R, CHANUTE
ABDOU, NABIH I, KANSAS CITY
ACEVEDO, ALFREDO, WICHITA
ADAMS, OWIGHT, OSAGE CITY
ADAMS JR, MARCUS W, HUTCHINSON
AGAN, LAWRENCE M, TOPEKA
AGUSTIN, CONRADO M, WICHITA
AHLSTRAND, RICHARD A, WICHITA
AILLON, ALEJANORO J, HALSTEAD
AKERS, GUY I, FORT SCOTT
AL-BAGHAL, MOHAMMAD, LEAVENWORTH
ALBERS, ROBERT C, HAYS
ALBRIGHT, JEROLD O, HUTCHINSON
ALDERSON, THOMAS W, GREAT BEND
ALDIS, HENRY, FORT SCOTT
ALDIS, WILLIAM, FORT SCOTT
ALDOROTY, NEIL, WICHITA
ALEXANDER, CHARLES E, KANSAS CITY
ALEXANDER, CLYDE W, KANSAS CITY
ALEXANDER, ELIZABETH, WICHITA
ALFONSO, MANUEL, WICHITA
ALGIE, WILLIAM H, KANSAS CITY
ALLBRITTEN JR, FRANK F, CUNNINGHAM
ALLEGRE, ANN, KANSAS CITY
ALLEN, FRANCES A, NEWTON
ALLEN, JAMES E, HAYS
ALLEN, MARK LYNN, KANSAS CITY,MO
ALLEN, MAX S, SHAWNEE MISSION
ALLEN, MONTE L, SALINA
ALLEN, PHILLIP M, WICHITA
ALLEN, RAY E, LIBERAL
ALLEN, TIMOTHY E, KANSAS CITY
ALLEN, WILLIAM R, KANSAS CITY
ALLEN JR, WILLIAM R, LEAVENWORTH
ALLRED, CHARLES T, COUNCIL GROVE
ALMONTE, PRISCILLA C, WICHITA
ALMONTE, RODOLFO O, WICHITA
ALQUIST, VERYL O, BAXTER SPRINGS
ALTENBERND, ELVIN CONRAD, SHAWNEE MISSION
ALVAREZ, LUIS A, SHAWNEE MISSION
ALVAREZ, NORBERTO, ARKANSAS CITY
AMARE, MAMMO, KANSAS CITY
AMAWI, MOHAMMAD S, DODGE CITY
AMBLER, CARL O, PRATT
AMEND, DOUGLAS J, EMPORIA
*AMMAR, ALEX D, WICHITA
ANDERSON, DALE W, AUGUSTA
ANDERSON, EUGENE G, WICHITA
ANDERSON, JODY, SALINA
ANDERSON, LARRY R, WELLINGTON
ANDERSON, LYLE B, BLOOMINGTON, IN
ANDERSON, SEVERT A, CLAY CENTER
ANDERSON, WILLIAM A, KANSAS CITY,MO
ANDERSON, WINSTAN L, LAWRENCE
ANDREASEN, RAYMOND L, COLUMBUS
ANTRIM, PHILIP JENIFER, ANTHONY
ANWAR, M ZIA, LEAVENWORTH
APPENFELLER, WILLIAM O, OSAWATOMIE
APPLEGATE JR, FRANCIS R, HAYS
ARAKAWA, KASUMI, KANSAS CITY
ARENAL, ANGELA C, KANSAS CITY
ARGO, DONALD A, MARYSVILLE
ARGOSINO, RODOLFO, WICHITA
ARMBRUSTER, ALBERT A, SHAWNEE MISSION
ARMSTRONG, HAROLD J, PITTSBURG
ARONOFF, MICHAEL E, OLATHE
ARREDONDO, MARIO, TOPEKA
ARROYO, ZEFERINO, GARDEN CITY
ARTMAN, JOHN C, HAYS
ARTZ, TYRONE D, WICHITA
ARTZER, DENNIS C, TOPEKA
ARUNAKUL, PUNYA, TOPEKA
ASBURY, LAWRENCE J, OLATHE
ASHEP, MARC A, KANSAS CITY
ASHKAR, ADNAN A, LEAVENWORTH
ASHLEY, BYRON J, TOPEKA
ASHLEY, SAMUEL G, CHANUTE
ASHLEY JR, B JOHN, TOPEKA
ASHMORE, ARTHUR L, WICHITA
ATHON, MERRILL D, SHAWNEE MISSION
ATKIN, JOHN O, YATES CENTER
ATKINS JR, FLOYD L, KANSAS CITY
ATLURU, NARAYANA RAO, TOPEKA
ATWOOD, M DALE, KINSLEY
AUCAR, ALFREDO, ARKANSAS CITY
AUCHARD, VIRGIL M, BUCYRUS,OH
AUNINS, JOHN, WICHITA
AUSTENFELD, MARK S, EMPORIA
AUSTIN, JOHN O, GARDEN CITY
AUSTIN, KENNETH D, GOODLAND
AVERILL, STUART C, TOPEKA
AVES, AGNES, PARSONS
AVES, RENATO B, PARSONS
AVILA, OSCAR A, DODGE CITY
AYUTHIA, ISSARA I, DODGE CITY

B

BACANI, OSWALDO, EFFINGHAM
*BACKES, DAVID J, WICHITA
BACON, ARTHUR H, LAKE WORTH,FL
BADEEN II, LOUIS JOHN, SHAWNEE MISSION
BAEHR, RALPH H, TOPEKA
BAEKE, JOHN O, SHAWNEE MISSION
BAILEY, COLIN, HALSTEAD
BAILEY, DONALD C, WICHITA
BAILEY, WILLIAM A, LAWRENCE
BAILIE, MICHAEL DAVID, KANSAS CITY
BAIR, ALBERT E, INDEPENDENCE
BAIR, GLENN D, TOPEKA
BAKER, FREDERICK C, TOPEKA
BAKER, HENRY K, CHANUTE
BAKER, PHILLIP L, TOPEKA
BAKER, RAY D, TOPEKA
BAKER, RICHARD B, MANHATTAN
BAKER, WILLIAM STEVEN, SHAWNEE MISSION
BAKER, ROBERT R, D.O., ARKANSAS CITY
BALANDFF, ARNOLD Z, SHAWNEE MISSION
BALL, RALPH G, MANHATTAN
*BAMBARA, JOHN F, MANHATTAN
*BAMMEL, BRUCE, WICHITA
BANKS, GILBERT, COFFEYVILLE
BANKS, ROBERT E, PAOLA
BANSAL, ROOPA O, SHAWNEE MISSION
BANSAL, SATISH C, SHAWNEE MISSION
BAPTIST, JEREMY E, SHAWNEE MISSION
BARABAN, MARC R, TOPEKA
BARBA, ESTRELLA G, WICHITA
BARBA JR, ANTONIO P, WICHITA
BARBER, JAMES L, AUGUSTA
BARBERA, PORTER E, INDEPENDENCE
BARE II, CHARLES E, SHAWNEE MISSION
BARKER, BENJAMIN W, WICHITA
BARKER, ELIZABETH B, SHAWNEE MISSION
BARKER, JAMES BERTON, SHAWNEE MISSION
BARKER, PATRICK N, PRATT
*BARKER, PATSY, WICHITA
BARKER, ROYAL A, COUNCIL GROVE
BARKER, STANTON L, HUTCHINSON
BARKER, STEVEN E, MINNEAPOLIS
BARLOW, JOHN M, MANHATTAN
BARNARD III, JAMES A, GARDEN CITY
BARNES, JOE L, WICHITA
BARNES, MARIAN, LANCASTER,CA
BARNETT, ARNOLD M, WICHITA
BARNETT, JAMES A, EMPORIA
BARNETT JR, THOMAS E, SHAWNEE MISSION
BARNHART, RONALD J, SHAWNEE MISSION
BARNHORST, DONALD A, SHAWNEE MISSION
BARR, RICHARD N, SHAWNEE MISSION
BARRICK, BRUCE, SHAWNEE MISSION
BARRY, DAVID R, LEAVENWORTH
BARTAL, ELY, WICHITA
BARTLETT, WAYNE C, WICHITA
BASCOM, CHARLES H, MANHATTAN
BASCOM, GEORGE S, MANHATTAN
BASER, ALI N, TRAVIS AFB,CA
BASHAM, JAMES J, FORT SCOTT
BASINGER, BRADLEY B, KANSAS CITY
BASS II, DRAL E, WICHITA
BASS JR, LEWIS N, KANSAS CITY
*BASSELL, G M, WICHITA
BASSETT, P MARCUS, HIAWATHA
BATES, MICHAEL O, WICHITA
BATES, MICHAEL NICHOLS, NEWTON
BATNITZKY, SOLOMON, KANSAS CITY
BATTISTE, CYNTHIA ELAINE, WICHITA
BATTY, LARRY H, SHAWNEE MISSION
BATTY, THOMAS V, SHAWNEE MISSION
BAUCOM, KARAN YVONNE, TOPEKA
BAUCOM-COPELAND, SHARON LAVARNE, TOPEKA
BAUDE, EUGENE L ANDRE, TOPEKA
BAUER, LAFE W, SHAWNEE MISSION
BAUER, MARTIN L, SHAWNEE MISSION
BAUER, THOMAS A, HUTCHINSON
BAUM, ARNOLD H, DODGE CITY
BAUMAN, M LEON, WICHITA
BAUMANN, PAUL A, WICHITA
BAXTER, KIRKMAN G, SHAWNEE MISSION
BAXTER, W REESE, SALINA
BAYLES, HUGH G, FREDONIA
BEACH, RICHARD R, TOPEKA
BEAHM, ANOL W, GREAT BEND
BEAHM, DONALD E, GREAT BEND
BEAHM, EDGAR H, INDEPENDENCE
BEAL, RAYMOND J, FREDONIA
BEALE, DAVID A, TOPEKA
BEAMON, RICHARD F, SHAWNEE MISSION
BEATY, EVERT C, PARSONS
BEATY, JAMES R, MAYETTA
BEAVER, JAMES L, WICHITA
BEBAK, DONALD M, WICHITA
BEBER, JORGE H, WICHITA
BECK, CHARLES W, WICHITA
BECK, JOSEPH O, TOPEKA
BECKER, KARL E, WICHITA
BECKER, LESLIE E, KANSAS CITY
BEDFORD, D R, TOPEKA
BEEBE, EDMER, OLATHE
BEELMAN, FLOYD C, TOPEKA
BEGGS, DAVID F, GARDEN CITY
BEHRHORST, CARROLL D, GUATEMALA,
BEIDERWELL, PAUL L, BELLEVILLE
BEJAR, JOSE M, KANSAS CITY
BELCHER, GEORGE D, COLUMBUS
BELL, DELORIS W, SHAWNEE MISSION
BELL, MARGARET E, APO NEW YORK,NY
BELLAR, RALPH E, HARPER
BELLER, WILLIS L, TOPEKA
BELOT JR, MONTI L, LAWRENCE
BELZER, EDWARD G, SHAWNEE MISSION
BENA, JAMES H, PITTSBURG
BENAGE, JOHN F, FORT SCOTT
BENNETT, CHARLES A, KANSAS CITY,MO
BENSON, KIRK T, KANSAS CITY
BENTON, JAY S, NEWTON
BERGIN, JAMES J, KANSAS CITY
BERKEY, VERNON A, PITTSBURG
BERKLEY, DON H, ABILENE
BERKLEY, NORMAN W, SENECA
BERLAND, DAVID I, TOPEKA
BERRY, JOHN M, SHAWNEE MISSION
BETHEL, CHANOLER S, WICHITA
BETTEGA, PAULO R, SHAWNEE MISSION
BETTERTON, DALE C, DODGE CITY
BEUGELSDIJK, HENRY PETER, HALSTEAD
BHARGAVA, ASHOK KUMAR, LA CROSSE
BHARGAVA, BAIKUNTH N, WICHITA
BHARGAVA, SHOBHANA, LA CROSSE
BICHLMEIER, FRANKLIN G, KANSAS CITY
*BICKELHAUPT, ETHAN E, TOPEKA
BIERI, PETER V, LAWRENCE
BIERLEIN, KENNETH J, PITTSBURG
BIERMANN, HENRY J, WICHITA
BIERMANN, WILLIAM J, WICHITA
BIGGS, DENNIS, ABILENE
BIGLER, F CALVIN, GARDEN CITY
BIKALES, VICTOR WILLIAM, SHAWNEE MISSION
BILLINGS, THOMAS, MCPHERSON
BILLINGSLEY, THAD H, KANSAS CITY
BINGAMAN, ROBERT W, WICHITA
BINYON, KERNIE W, WICHITA
BISHOP, FRANCIS E, SHAWNEE MISSION
BISHOP, ROONEY LEE, LAWRENCE
BITTENBENDER, LEE R, LAWRENCE
BITZER, DONALD A, WASHINGTON
BIXLER II, THOMAS J, KANSAS CITY
BLACK, CYRIL V, PRATT
BLACK, WILLIAM L, FT CARSON, CO
BLACKBURN, ROBERT W, COUNCIL GROVE
BLAIR, T RICHARD, LAWRENCE
BLAKE, HENRY S, TOPEKA
BLANK, JOHN N, HUTCHINSON
BLANKENSHIP, JIM O, OTTAWA
BLAYLOCK, HOYT C, WICHITA
BLETZ, DONALD B, SHAWNEE MISSION
BLISS, JOY V, OLATHE
BLOOD, MARY J, WICHITA
BLOOM, L THEIL, KINGMAN
*BLOOM, RODNEY LAMONT, WICHITA
BLOXHAM, THOMAS J, WICHITA
BLUM, MICHAEL A, D.O., OLATHE
BODENSTEINER, DAVID C, KANSAS CITY
BOESE, KENNETH M, MANHATTAN
BOGGAN, MICHAEL O, KANSAS CITY
BOGNER, PAUL F, CLAY CENTER
BOLES, J MICHAEL, SHAWNEE MISSION
BOLES, R DALE, DODGE CITY
BOLINGER, ROBERT E, KANSAS CITY
BOLLMAN, CHARLES S, JUNCTION CITY
BOLT, MICHAEL, NEWTON
BOLTON, VICTOR E, KANSAS CITY
BOND, ROGER C, WICHITA
BONEBRAKE, C RICHARD, TOPEKA
BOREL, DAVID, TOPEKA
BORGENDALE, LLEWELLYN V, WAMEGO
BORRA, MARIO J, HUTCHINSON
BOS, NORMAN C, HUTCHINSON
BOSILEVAC, FRED N, KANSAS CITY
BOSILJEVAC JR, JOSEPH E, EMPORIA
BOSSE, FRANK K, ATCHISON
BOSTON, GARY O, LEAVENWORTH
BOUDA, DAVID W, HUTCHINSON
BOUDET, ROBERT A, KANSAS CITY,MO
BOWEN, CLOVIS W, TOPEKA
BOWEN JR, HARRY J, TOPEKA
BOWEN, JAMES D, ASHLAND
BOWMAN, ANTHONY J, WICHITA
BOYD, SPENCER H, TOPEKA
BOYD, Z REX, WICHITA
BOYDEN, MARY S, LAWRENCE
BOYER, ROBERT E, KINGMAN
BOYLE, HUGH H, WICHITA
BRACKETT JR, CHARLES E, KANSAS CITY
BRADA, DONALD ROBERT, HUTCHINSON
BRADEN, BILL L, WAMEGO
BRADLEY, H RUSSELL, EMPORIA
BRADLEY, J RODERICK, GREENSBURG
BRADY, CHARLES S, ATCHISON
BRAHMAN, HERBERT D, TOPEKA
BRAKE, DAVID, WICHITA
BRANDSTED, ERNEST C, MCPHERSON
BRANSON, VERNON L, LAWRENCE
BRAUN, EDWARD W, FORT SCOTT
BRAUN, KENNETH, WICHITA
BRAUN, ROBERT W, TOPEKA
BRAUN, THOMAS G, WICHITA
BRAUN, WILLIAM T, PORT ORANGE,FL
BRAUN III, WILLIAM T, WICHITA
BRAUNSDORF, ROBERT L, TOPEKA
BRAVERMAN, DAVID ELLIOTT, SHAWNEE MISSION
BRAY, AVIS PAGE, CONCORDIA

* Probationary members.

BRECKBILL, DAVID L, WICHITA
BRENNER, WILLIAM R, LARNED
BRETHOUR, LESLIE J, JUNCTION CITY
BREWER, MARSHALL A, ULYSSES
BRIAN, ROBERT M, EL DORADO
BRIBACH, EUGENE J, ATCHISON
BRIDGENS, JAMES G, SHAWNEE MISSION
BRIDWELL, RUSSELL E, TOPEKA
BRILLHART, MAXINE T, KANSAS CITY
BRINTON, E HOLMES, WICHITA
BRINTON, EDWARD S, WICHITA
BRITO, RAUL E, WICHITA
BROCKHOUSE, JOHN P, EMPORIA
BROOKER, ROBERT M, SANTA BARBARA,CA
BROOKS, WILLIAM HENRY, KANSAS CITY
BROSIUS, FRANK C, WICHITA
BROUCEK, FRANCIS J, SHAWNEE MISSION
BROWN, ALEX L, CLEARWATER,FL
BROWN, C EVERETT, STAFFORD
BROWN, DAVID J, WICHITA
BROWN, FRED E, ST MARYS
BROWN, JOHN V, KANSAS CITY
BROWN, MICHAEL P, WICHITA
BROWN, PAUL W, OLATHE
BROWN, RANDALL J, WICHITA
BROWN, ROBERT L, WICHITA
BROWN, ROBERT M, MANHATTAN
BROWN, ROBERT O, ATCHISON
BROWN, ROBERT WAYNE, SALINA
BROWN, RONALD C, WICHITA
BROWN, RONALD L, WICHITA
BROWN, VAL J, WICHITA
BROWN, WILLIAM P, SHAWNEE MISSION
*BROWN JR, VAL J, WICHITA
BROWN-SANDERS, CAROLINE, LEES SUMMIT,MO
BROWNING, JIMMIE L, COTTONWOOD FALS
BROWNING, WILLIAM H, WICHITA
BROWNING, WILLIAM R, MADISON
BROWNRIGG, RICHARD L, DODGE CITY
BROXTERMAN, STEVEN JOSEPH, SHAWNEE MISSION
BRUMMETT, RICHARD R, SALINA
BRUNER JR, KENNETH W, WICHITA
BRUNFELDT, JOAN KRAUS, LAWRENCE
BRUNGARDT, BERNARD A, SALINA
BRUNING, ROGER MARION, SHAWNEE MISSION
BRUNO, JAMES W, GARDEN CITY
BRYAN, EMERY C, ERIE
BUBB, STEPHEN K, KANSAS CITY
BUBECK, RALPH W, WICHITA
BUCK JR, BEN H, WICHITA
BUCK JR, HENRY W, LAWRENCE
BUCKMAN, MARTIN SPALDING, SHAWNEE MISSION
BUCKRIDAN, LATIF, WICHITA
BUDETTI, JOSEPH A, N MIAMI BEACH,FL
BUESING, OLIVER R, LEAVENWORTH
BULA, RALPH E, HAYS
BUNKER JR, HERBERT L, JUNCTION CITY
BURDICK, BRUCE M, MANHATTAN
BURDZIK, EBERHARD G, TOPEKA
BURGER, J DALE, HUTCHINSON
BURGER, PAUL B, SHAWNEE MISSION
BURGER, WILLIAM E, KANSAS CITY
BURGESON, FRANK G, EMPORIA
BURGESS, ARTHUR P, OSWEGO
BURKE, JAMES J, FORT SCOTT
BURKE, JOSEPH V, ATCHISON
BURKET JR, GEORGE E, KINGMAN
BURKMAN, REUBEN J, CHANUTE
BURNETT, A DEAN, HALSTEAD
BURNEY, WILLIAM W, WICHITA
OURNEY II, WILLIAM W, WICHITA
BURPEE, JAMES F, WICHITA
BUSCH, ANTHONY B, DODGE CITY
BUSTOS, JONAS G, HERINGTON
BUTCHER, THOMAS P, EMPORIA
BUTH, DENNIS K, WICHITA
BUTIN, J WALKER, WICHITA
BUTIN, RICHARD E, CLEVELAND,OH
BUTLER, DORIS C, WICHITA
OUTT, MUHAMMAD, CONCORDIA
BUTTERFIELD, MARI ANNE, KANSAS CITY
BYERS, MARTHA S, KANSAS CITY
BYRNE, JAMES PERRY, WICHITA

C

CABRERA, ALBERTO, ELKHART
CACHIA, RICHARD M, TOPEKA
CAEDO, CARMELITA D, LIBERAL
CAING, MATILDE V, PARSONS
CALBECK, JOHN, GARDEN CITY
CALDERON, JAIME, KANSAS CITY
CALIENDO JR, DANIEL J, WICHITA
CALKINS, JOHN W, SHAWNEE MISSION
CALKINS, LARRY L, SHAWNEE MISSION
CALKINS, W GRAHAM, KANSAS CITY,MO
CAMERON, WILLIAM J, KANSAS CITY
CAMPBELL, EDWARD G, EMPORIA
CAMPBELL, FRANCES S, NEWTON
CAMPBELL, GARLAND L, ARKANSAS CITY
CAMPBELL, WILLIAM H, COFFEYVILLE
CAMPION, WOODROW M, LIBERAL
*CANNON, MICHAEL W, WICHITA
CAPPER, STANLEY L, WICHITA
CAPOUFF, JAY J, SHAWNEE MISSION

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CAREY, LARRY J, PARSONS
CARLETON, RICHARD C, CLAY CENTER
CARLIN, JAMES WILLARD, KANSAS CITY
CARLSON, EARL V, HAYS
CARLSON, TERRY S, WICHITA
CARNAHAN, ROBERT L, LAWRENCE
CARPENTER, PAUL R, KANSAS CITY
CAPPER, IVAN H, NEWTON
CARPER, OWEN E, NEWTON
CARPASCO, LENOR C, SHAWNEE MISSION
CARREAU, ERNEST P, CEDRIDGE,CO
CARPO, F AURELIO, WINFIELD
CARRO, TONY L, KANSAS CITY
CARSON, BERTRAM H, D.O., WICHITA
CARTER, MACK A, WICHITA
CARTY, RUSSEL W, SHAWNEE MISSION
CASEY, JAMES, HUTCHINSON
CASEY, JOHN J, WICHITA
CASHMAN JR, MAURICE R, TOPEKA
CASTEEL, CHARLES K, SHAWNEE MISSION
CATHEY, ROBERT H, MANHATTAN
CATTANEO, ERNEST A, SHAWNEE MISSION
CAUBLE, WILBUR G, WICHITA
CAVANAUGH, CLAIR J, GREAT BEND
CAVANAUGH, JOHN W, TOPEKA
CAVITT, ROBERT F, SHAWNEE MISSION
CAWLEY, LEO P, WICHITA
CECIL III, JOHN, HAYS
CEDERLIND, CRANSTON JAY, SHAWNEE MISSION
CENAC, MARK T, LEAVENWORTH
CHAFFEE, DEAN C, ABILENE
CHAFFIN JR, GOODLOE S, PRATT
CHALIAN, ALEXANDER R, KANSAS CITY
CHAMBERLIN JR, CECIL R, TOPEKA
CHANEY, ERNIE J, WICHITA
CHANG, C H JOSEPH, KANSAS CITY
CHANG, FREDERIC C, WICHITA
CHANG, SHU FANG, SHAWNEE MISSION
CHAPMAN, JAMES H, WICHITA
CHAPPUIE, WILLIAM G, INDEPENDENCE
CHARO, FREDERICK H, WICHITA
CHAVALA, SUDARSAN, FT LEAVENWORTH
CHEDIAK, ELIAS, LAWRENCE
CHEN, CHU-CHI, TOPEKA
CHEN, TAK-MING, TOPEKA
CHENOWETH, JOHN R, D.O., OLATHE
CHEPRY JR, ARTHUR C, TOPEKA
CHEPVEN, PHILIP L, HUTCHINSON
CHI, IL-SUNG, WICHITA
CHIN, TOM O, KANSAS CITY
CHO, CHENG T, KANSAS CITY
CHO, SECHIN, WICHITA
CHONKO, ARNOLD M, KANSAS CITY
CHOPRA, PAMAN, WICHITA
CHOTIMONGKOL, ANUPONG, DODGE CITY
CHOW, STANLEY Y, FORT SCOTT
CHOY, JAMES K L, SUN CITY,AZ
CHPISTENSEN, MARION O, KIOWA
CHRISTENSEN, SHANE R, KANSAS CITY, MO
CHPISTIAN, STANLEY J, SHAWNEE MISSION
CHPONISTER, BERT, NEODESHA
CHRYSANT, STEVEN G, KANSAS CITY
CHUBB, RICHARD M, BAXTER SPRINGS
CHUNG, JOHN J, SHARON SPRINGS
CISKEY, WILLIAM J, EUREKA
CLAASSEN, MILTON A, NEWTON
CLARK, COURTNEY, WICHITA
CLARK, CRAIG N, TOPEKA
CLARK, DAVID H, SALINA
CLARK, LAURENCE A, WAMEGO
CLARK, ORVILLE R, ST PETERSBURG,FL
CLARK, RAY A, LAKE CHAS,LA
CLARK, ROBERT G, WICHITA
CLARK, ROBERT THOMAS, GUATEMALA CEN A,
CLASSEN, SAMUEL D, MCPHERSON
CLENDENIN, ROBERT KEELE, SHAWNEE MISSION
CLIFTON, H DAVID, WICHITA
CLINE, BYRON W, WICHITA
CLINTON, DALE L, LAWRENCE
CLYMER, DAVID J, SHAWNEE MISSION
COALE, LLOYD H, KANSAS CITY
COBB, LESLIE H, MULVANE
COCHRAN, PAUL W, TOPEKA
CODY, DOROTHY, HAYS
CODY, JOHN, HAYS
COE, RICHARD D, SHAWNEE MISSION
COFFEY, ROY B, SALINA
COHEN, JUSTIN THOMAS, WICHITA
COHEN, LOUIS, TOPEKA
COHEN, MARC O, SHAWNEE MISSION
COHEN, ROBERT A, SHAWNEE MISSION
COHLMIA, JERRY D, WICHITA
COHN, STEVEN G, SHAWNEE MISSION
COHNBERG, ROSELLEN E, CEDAR VALE
COKELEY, JOHN M, TOPEKA
COKER, W LAURENCE, TOPEKA
COLDSMITH, DONALD C, EMPORIA
COLE, WARD M, WELLINGTON
COLEMAN, GARY, ABILENE
COLEMAN, ROBERT L, SHAWNEE MISSION
COLEMAN, THOMAS J, WICHITA
COLIP, F MERLYNN, NORTON
COLLIER, HAROLD W, WICHITA
COLLIER, WILLIAM J, MCPHERSON
COLLINS, DEAN T, TOPEKA
COLLINS, EDWARD JOSEPH, TOPEKA
COLLINS, ELISABETH P, TOPEKA
COLLINS, FRANCIS T, TOPEKA
COLLINS, SHARON A, GARDEN CITY
COMBS, PETER S, LEAVENWORTH

CONARD, CLAIR C, DODGE CITY
CONCEPCION JR, EUGENIO S, WICHITA
CONNELLY, MAURICE R, SALINA
CONNER, BRIAN, SALINA
CONRARDY, PETER A, WICHITA
COOK, DONALD PAY, WICHITA
COOK, G EDWARD, WICHITA
COOK, JAMES O, KANSAS CITY
COOKE, ALLAN R, KANSAS CITY
COOLEY, DAVID A, SHAWNEE MISSION
COOLEY, DENNIS M, TOPEKA
COOMER, TYLER E, PITTSBURG
COONFIELD, JAMES W, KANSAS CITY
COOPER, ARTHUR E, NORTON
COOPER, JACK R, SHAWNEE MISSION
COOPER, KENT J, PITTSBURG
COOPER, LEO F, DREXEL,MO
*COOPER, MICHAEL K, WICHITA
COPELAND, GARY A, JUNCTION CITY
COPENING, TELL B, IOLA
CORBIN, MURRAY D, SHAWNEE MISSION
CORDER, ROBERT L, ST JOSEPH,MO
CORDER, S SCOTT, OTTAWA
COSSETTE, JERROLD E, SALINA
COSSMAN, F PRICE, WICHITA
COTTON, ROBERT T, TOPEKA
COULTER, HENRY F, SHAWNEE MISSION
COULTER, THOMAS B, SHAWNEE MISSION
COULTER, THAYNE A, D.O., CONCORDIA
COWLES, GORDON T, WICHITA
COX, ROBERT H, HAYS
COX III, IRA L, KANSAS CITY
COX JR, IRA, SHAWNEE MISSION
COX JR, WALLACE F, KANSAS CITY
COYLE, JOHN F, COFFEYVILLE
CRABB, JEREMIAH E, KANSAS CITY, MO
CRAIG, CHARLES C, NEWTON
CRAIG, THOMAS A, JUNCTION CITY
CRAM, ERNEST R, ST FRANCIS
CRAM JR, OLE R, LARNED
CRAMER, GUY W, PARSONS
CRAMM, RUSSELL E, HAYS
CRANDALL, DORA, ATTICA
CRANE, C HERBERT, MANHATTAN
CRANE, DAVID O, WICHITA
CRANSTON, STEPHEN D, NEWTON
CRARY, JOHN E, TOPEKA
CROCKETT, CHARLES A, KANSAS CITY
CRONIN, DONALD J, WICHITA
CROSS, BETTY S, KANSAS CITY
CROUCH, STEVEN W, TOPEKA
CROUCH, WILLIAM H, TOPEKA
CROW, ERNEST W, WICHITA
CROWLEY, EDWARD X, WICHITA
CULP, LOUIS M, KANSAS CITY
CULTRON, FRANK T, SALINA
CULVER, WARREN T, LAWRENCE
CUMMINGS, RICHARD J, WICHITA
CURA, ELSA C, MONTEZUMA
CURRAN, KEVIN E, SHAWNEE MISSION
CURTIS, MIRJANA R, KANSAS CITY
CZAPANSKY, DESIREE K, KANSAS CITY

D

D'SOUZA, BISMARCK C, SALINA
DAEHNKE, SIGURD S, WINFIELD
DAHL, ASHER W, COLBY
DAILEY, RONALD F, JOHNSON
DAIZ, ANTONIO S, PARSONS
DAKHIL, SHAKER R, WICHITA
DALUM, PETER JOSEPH, CLAY CENTER
DANIELS, ROBERT M, VALLEY CENTER
DARR, RICHARD B, KANSAS CITY
DARRAH, CAROL J N, WICHITA
DAVIDSON, HARRY T, WICHITA
DAVIS, CHESTER R, TOPEKA
DAVIS, CHRISTOPHER G, KANSAS CITY
DAVIS, DAVID H, LARNED
DAVIS, DAVID R, EMPORIA
DAVIS, GEORGE R, ELLSWORTH
DAVIS, PAUL H, WICHITA
DAVIS, RICHARD E, SHAWNEE MISSION
DAVIS, RONALD B, WICHITA
DAVIS JR, JAMES W, KANSAS CITY,MO
DAY, HOWARD, WICHITA
DAY, HUGHES W, KANSAS CITY
DE BAKKER, JAN B, WICHITA
DE HART, ARTHUR DONIVA, WICHITA
DE SMET, ARTHUR AUGUST, KANSAS CITY
DE SOUZA, DERRICK J, LEAVENWORTH
DE VRIES, PIETER A, KANSAS CITY
DECENA, IMMACULADA, LEAVENWORTH
DECHAIRO, THOMAS, WESTMORELAND
DECKER, DONALD D, HALSTEAD
DEGNER, JAMES B, GREAT BEND
DEITZ, MICHAEL R, KANSAS CITY
DEJONG, DAVID C, WICHITA
DELCORE, POMANO, TOPEKA
DELGADO, SERGIO, TOPEKA
DELLETT, KENNETH B, EL DORADO
DELP, MAHLON H, SHAWNEE MISSION
DELPHIA, ROBERT E, OLATHE
DEMOSS, ELEANOR P, WICHITA
DEMOTT, WAYNE R, KANSAS CITY

DENISON, TERRY R, SHAWNEE MISSION
DENNING, DALE P, WICHITA
DEPENBUSCH, FRANCIS L, HUTCHINSON
DERRINGTON, KENNETH L, SHAWNEE MISSION
DESOIGNIE, RAFAEL R, TOPEKA
DETAR, GEORGE F, IOLA
DIACON, JAMES L, WICHITA
DIALLO, GASTON I, KANSAS CITY
*DIAZ, DOLORES M, WICHITA
*DIAZ, SALVADOR F, WICHITA
DICK, ARTHUR R, KANSAS CITY
DICK, WILLIS G, IOLA
DICK JR, HENRY J, CHANUTE
DICKINSON, CHARLES R, COFFEYVILLE
DIEDERICH, DENNIS A, KANSAS CITY
DIEHL, ANTONI M, KANSAS CITY, MO
DIENER, CLAYTON H, HESSTON
DILL, RODNEY, ATWOOD
DILLON, STEVEN C, PRATT
DILLON, WILLIAM L, PARSONS
DIRKSEN, HANS C, WICHITA
DIXON, RAYMOND W, COFFEYVILLE
DOBRATZ, ROBERT A, BELOIT
DOCKHORN, ROBERT J, SHAWNEE MISSION
DOEBLIN, P LAURENCE, WICHITA
DOHERTY, WILLIAM R, SHAWNEE MISSION
DOLAN JR, PHILIP JARVIS, WICHITA
DONATELLE, EDWARD P, WICHITA
DONLEY, JAMES L, WICHITA
DONNELL, JAMES M, WICHITA
DOORNBOS, J FRED, WICHITA
DORSCH, JOHN N, HAYS
DOUBEK, HERBERT D, BELLEVILLE
DOUGHERTY, THOMAS M, GARNETT
DOUGLAS, JOSEPH MAHLO, LAWRENCE
DOWELL, JAMES C, SALINA
DOZIER, FRED S, HERINGTON
DRAEMEL, H RICHARD, SALINA
DRAKE, DOUGLAS J, BELOIT
DRAKE, RALPH L, WICHITA
DRANGE, ROBERT K, WICHITA
DRAZEK, GEORGE, WICHITA
DREHER, HENRY S, SALINA
DREVETS, CURTIS C, WICHITA
DUCKETT, THOMAS G, HIAWATHA
DUCKETT II, THOMAS G, SHAWNEE MISSION
DUHON, JOHN MARQUE, HUTCHINSON
DUICK, GREGORY, WICHITA
DUJOVNE, CARLOS A, KANSAS CITY
DULIN, JOSE I, KANSAS CITY
DUNAGIN, JACK A, TOPEKA
DUNIVEN, PHILIP L, TOPEKA
DUNLAP, MICHAEL O, ATCHISON
DUNLAP, RICHARD L, LAWRENCE
DUNN, DANIEL R, SCOTT CITY
DUNN, MARVIN I, KANSAS CITY
DUNSHEE, CARLYLE M, FORT SCOTT
DUNSHEE, CHERYL A, WICHITA
DURAND, ANTONIO C, WICHITA
DURKEE, WILLIAM R, MANHATTAN
DURST JR, ROBERT D, TOPEKA
DUYSAK, SAMI, LEAVENWORTH
DYCK, ARTHUR H, MCPHERSON
DYCK, CORA E, WICHITA
DYCK, ERIC LEE, HAYS
DYCK, GEORGE, NEWTON
DYER, VERNON E, WICHITA
DYSART, JACK C, STERLING

E

EASTES, GARY DEAN, HALSTEAD
EATON, EDWARD L, TOPEKA
EATON, GLEN E, SALINA
EATON, LESLIE F, SALINA
ECKART, DE MERLE E, HUTCHINSON
ECKERT, WILLIAM G, WICHITA
EDDY, VICTOR M, HAYS
EDROZO, M LUZ LUNA, COFFEYVILLE
EDWARDS, DAVID J, EMPORIA
EDWARDS, MANIS C, WICHITA
EGEA, FERNANDO M, KANSAS CITY
EGELHOF, RICHARD H, WICHITA
EICHHORN, FRANK D, GARDEN CITY
EIDT, DAVID W, OLATHE
EIDT, LAURENCE A, OLATHE
EIKERMANN, WILLIAM C, SHAWNEE MISSION
ELDER, DOUGLAS M, TOPEKA
ELDREDGE, LOUIS D, RUSSELL
ELLEDGE, E FRED, GREAT BEND
ELLIS, BOBBY J, EMPORIA
ELLIS, HARVEY D, WICHITA
*ELLIS, JOSEPH G, WICHITA
ELLIS, STEPHEN S, COFFEYVILLE
ELLISON, PAUL D, SALINA
ELNEN, WALTER T, WICHITA
EMPSON, CHARLES L, INDEPENDENCE
ENBERG, ROBERT, HAYS
ENDERS, WRAY, SHAWNEE MISSION
ENNS, EUGENE K, NEWTON
ENNS, JAMES H, LAKE HAVASU CTY, AZ
ENOCH, ROLLAND, WICHITA
ENS, GERHARD GEORGE, HILLSBORO
ENS, PETER, HILLSBORO

* Probationary members.

EPLEE, JOHN R, ATCHISON
ERICKSON, CLARENCE W, PITTSBURG
ERKEN, RONALD V, WICHITA
ERNST, R L, WICHITA
ESCH, JOHN G, PITTSBURG
ESRIG, HAROLD L, D.O., SHAWNEE MISSION
*ESTEP, THOMAS H, WICHITA
ESTRADA, EDMUNDO C, LIBERAL
ESTRADA, LINA, LIBERAL
ETZENHOUSER III, RUSSELL D, SHAWNEE MISSION
EULBERG, JEANNE G, KANSAS CITY
EVANS, CAROL ANN, SHAWNEE MISSION
EVANS, FARRIS D, WICHITA
EVANS, GRANT E, WICHITA
EVANS, JOHN F, WICHITA
EVANS, RICHARD W, MILWAUKEE, WI
EVANS, ROGER WILLIAMS, WICHITA
EVANS, WILLIAM R, GREAT BEND
EVANS JR, WILLIAM E, SHAWNEE MISSION
EWING, THOMAS O, LARNED
EYSTER, ROBERT L, WICHITA

F

FABIAN, CAROL J, KANSAS CITY
FAIRCHILD, JOHN A, MANHATTAN
FAIRCHILD, RICHARD S, TOPEKA
FALLON, JOHN H, KANSAS CITY, MO
FALTER, RICHARD T, HUTCHINSON
FARHA, GEORGE J, WICHITA
FARHA, S JIM, WICHITA
*FARLEY, JAMES A, WICHITA
FARRIS, RONNIE S, EMPORIA
FAST, ROBERT E, ATCHISON
FAST, W SPENCER, ATCHISON
FEAGAN, JERRY, TOPEKA
FEIGHNY, ROBERT E, SALINA
FELDMEYER, SEELEY T, SYRACUSE
FENDER JR, THOMAS H, WICHITA
FENT, LEE S, NEWTON
FENTON, ROBERT M, GARDEN CITY
FERGUSON, ROBERT LEON, SHAWNEE MISSION
FERNANDEZ, HECTOR O, HOISINGTON
FERNANDEZ, LUIS A, TOPEKA
FERNIE, ROBERT W, BOULDER, CO
FERREE, RICHARD ALLAN, MCPHERSON
FERRELL, DONALD P, WICHITA
FERRIS, BRUCE G, WICHITA
FESTOFF, BARRY W, KANSAS CITY, MO
FEUILLE JR, EDMOND G, WICHITA
FEVURLY, CHRIS D, LEAVENWORTH
FIELD, RICHARD A, TOPEKA
FIELDS, GALEN W, MCPHERSON
FIELDS, STEPHEN, D.O., WICHITA
FIELDSTONE, PAUL R, JUNCTION CITY
FIESER, CARL W, GREAT BEND
FILLEY, VERNON W, PRATT
FILLMAN, ELDON M, TOPEKA
FINK, ABRAHAM A, PLANTATION, FL
FINLEY, DENNIS R, WICHITA
FISCHER, REX R, MANHATTAN
FISHER, JAMES B, WICHITA
FISHER, RAY F, WICHITA
FISHER, RONALD M, MANHATTAN
FITZGERALD, E JAMES, GOODLAND
FITZGERALD, EDWARD J, WICHITA
FITZIG, SANFORD, WICHITA
FITZPATRICK, M ROBERT, KANSAS CITY
FLANDERS, H ALDEN, BOULDER, CO
FLECKENSTEIN, CHARLES S, ONAGA
FLEMING, FORNEY W, WICHITA
FLESKE, LEONARD T, GREAT BEND
FLOERSCH, HUBERT M, KANSAS CITY
FLOWERS JR, CLELL B, WICHITA
FORD, CHARLES R, WICHITA
FORD, FRED L, TOPEKA
FOROYCE, NORMAN, SHAWNEE MISSION
FORET, JOHN D, KANSAS CITY
FORRED JR, WALTER A, HARPER
FORSTER JR, LOUIS G, SALINA
FORTUNE, CEDRIC B, OLATHE
FOSS, DANIEL C, HUTCHINSON
FOSTER, D BERNARD, TOPEKA
FOWLER, DENNIS L, WINFIELD
FOWLER, ROBERT J, WICHITA
FOWLER, WAYNE L, CONCORDIA
FOX, DEANNA K, KANSAS CITY
FOX, HOWARD A, KANSAS CITY
FRANCIS, ANTHONY E, SALINA
FRANCIS, NORTON L, WICHITA
FRANCISCO, CLARENCE L, SHAWNEE MISSION
FRANCISCO, DAN A, WICHITA
FRANCISCO, EDGARDO, HORTON
FRANCISCO, LINDA L, WICHITA
FRANCISCO, W DAVID, KANSAS CITY
FRANSEN, HERBERT, NEWTON
FRANSEN, PAUL H, NEWTON
FRANZ, ROBERT G, HILLSBORO
FRASER, CRYSTAL A, WICHITA
FRAZIER, RICHARD L, EMPORIA
FREDERICK, M F, HUGOTON
FREDRICKSON, DUANE E, LINDSBORG
FREEBORN JR, WARREN S, CLYDE
FREEMAN, F GILES, PRATT
FREEMAN, FRED A, MANHATTAN
FREEMAN, MALCOLM C, PITTSBURG
FREEMAN, RAYMOND S, SALINA
*FRENCH, JEROME E, WICHITA

FRIESEN, DALE, LAWRENCE
FRIESEN, FLORENCE V, HESSTON
FRIESEN, ORLANDO J, BUHLER
FRIESEN, STANLEY R, KANSAS CITY
FRIGGERI, ROBERT W, GIRARD
FRITZ, GEORGE E, WICHITA
FRITZEMEIER, WILLIAM H, WICHITA
FROMER, JOEL, WICHITA
FROMM, ARTHUR H, WICHITA
FRY, LUTHER L, GARDEN CITY
FULLEN, JERYL G, SHAWNEE MISSION
FULLER, DERYL D, LINDSBORG
FULTON, JOHN K, WICHITA
FUNK, EDWARD D, BONNER SPRINGS
FUSILLO, MICHAEL, TOPEKA

G

GALICHIA, JOSEPH P, WICHITA
GALVAN, ALONSO, WICHITA
GANDHI, SHANTIKUMAR K, TOPEKA
GANN, E LAMONTE, EMPORIA
GANS, FREDERICK A, SALINA
GANZARAIN, RAMON C, TOPEKA
GARCIA, FRANCISCO, SHAWNEE MISSION
GARCIA, GOULD C, EMPORIA
GARCIA, GUILLERMO O, COOGE CITY
GARD, RAYMOND F, PRATT
GARDINER, TED M, GARDEN CITY
GARDNER, BILLIE L, HARPER
GARDNER, GLENN M, SHAWNEE MISSION
GARDNER, JAMES D, MANHATTAN
GAREY, WILLIAM JOHN, CHANUTE
GATENO, JOSEPH, GREAT BEND
GAY, JOHN D, TOPEKA
GEHRT, EARL B, CHANUTE
GEITZ, JAMES M, EMPORIA
GELVIN, E RAYMOND, CONCORDIA
GENCH, RAYMOND L, CARMEL, CA
GENDEL, JOSEPH E, TOPEKA
GENILO, AMANCIO C, WICHITA
GENILO, CELESTE A, WICHITA
GENTRY, KALE C, SHAWNEE MISSION
GEORGE, EARL F, WICHITA
GEORGE, M DON, WICHITA
GERBER, ALLEN O, WICHITA
GERBER, HARRY A, LEAVENWORTH
GERJARUSAK, PRAPAS, KANSAS CITY
GESSLER, DONALD J, WICHITA
GETTLER, DEAN T, FORT SCOTT
GIBBS, EUGENE, COFFEYVILLE
GIESSEL, MICHAEL O, TOPEKA
GILBERT, J HOWARD, SENECA
GILBERT, ROBERTA M, SHAWNEE MISSION
GILBERT II, JOHN H, GARDEN CITY
GILHOUSEN, FREDERIC M, KANSAS CITY
GILL, GEORGE L, LAMPE, MO
GILLEN, BILLY A, SHAWNEE MISSION
GILLES, HELEN M, LAWRENCE
GILMARTIN, RICHARD C, WICHITA
GIMPLE, KENNETH, TOPEKA
GINAVAN, DUANE A, EMPORIA
GIROD, CHARLES I, EL DORADO
GIRON JR, LOUIS T, KANSAS CITY
GIVNER, DAVID, WICHITA
GLATTER, THOMAS R, KANSAS CITY, MO
GLAZZARD, CHARLES D, OLATHE
GLEASON, JIMMIE A, TOPEKA
GLENN, JAMES N, EMPORIA
GLENN, LYLE G, PROTECTION
GLOERSEN, PETER F, SEVERNA PARK, MD
GLOVER, RICHARD M, NEWTON
GNAU, FREDRIC B, HALSTEAD
GODFREY, KENNETH E, TOPEKA
GODFREY, ROBERT G, KANSAS CITY, MO
GODFREY, WILLIAM A, KANSAS CITY
GODWIN, PHILLIP A, LAWRENCE
GOERING, DONALD D, COLDWATER
GOERING, EMIL L, BURLINGAME
GOERTZ, KENNETH K, KANSAS CITY
GOERTZ, LEO R, KANSAS CITY
GOHIL, MAHENDRA N, WICHITA
GOLDBERG, HERBERT R, WICHITA
GOLDSTEIN, GERALD L, SHAWNEE MISSION
GOLLERKERI, MOHAN P, SHAWNEE MISSION
GOLLIER, ROBERT A, OTTAWA
GOLLIER II, ROBERT A, OTTAWA
GOMETZ, MODESTO S, PITTSBURG
GOMEZ, FRANCISCO, SHAWNEE MISSION
GONZALEZ, HIRAM, WICHITA
GOOD, JAMES T, FORT SCOTT
GOOD, MICHAEL W, CLAY CENTER
GOOD, WENDELL LISLE, SHAWNEE MISSION
GOODPASTURE, HEWITT C, WICHITA
GOODPASTURE, WILLARD C, HUTCHINSON
GOODWIN, DONALD W, KANSAS CITY
GOODWIN, MARY K, ANDALE
GOOTEE, JOSEPH E, TOPEKA
GORDON, JAMES R, WICHITA
GOSALIA, ANIL V, SHAWNEE MISSION
GOTO, HIROSHI, KANSAS CITY
GOYLE, KRISHAN K, WICHITA
GOYLE, VIMAL, WICHITA
GRABER, CHARLES, NEWTON
GRADY, KENNETH L, KANSAS CITY
GRAHAM, KENNETH L, LEAVENWORTH
GRAHAM, SUSAN B, SHAWNEE MISSION
GRAHAM, THOMAS W, LEAVENWORTH

GRAHAM JR, CHARLES P, TOPEKA
GRANT, MICHAEL O, CONCORDIA
GRANTHAM, JAREO J, KANSAS CITY
GRAUEL, CHARLES W, WICHITA
GRAVES, JACK W, WICHITA
GRAVES, KATHRYN, HUTCHINSON
GRAY, C K, LAWRENCE
GRAY, C LUCIEN, WICHITA
GRAY, OAVIO E, TOPEKA
GRAY, H TOM, WICHITA
GRAYIR, ANTOINE S, TOPEKA
GRAYSON, ROY O, ALBUQUERQUE,NM
GREEN, LAWRENCE C, ARKANSAS CITY
GREENBERG, GEORGE E, DOOGE CITY
GREENBERG, MARK, TOPEKA
GREENBERGER, N J, KANSAS CITY
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GREENWOOD, EOWARD O, TOPEKA
GREENWOOD, JAMES F, GAROEN CITY
GREER, JAMES A, WICHITA
GREER, RICHARO H, TOPEKA
GREGORY, RALPH W, SHAWNEE MISSIDN
GRIBBLE, ROBERT N, WICHITA
GRIFFITH, FRANK H, SALINA
GRILLOT, FLOYO B, WICHITA
GRIMALOI, GARY A, FORT SCOTT
GRIMES, I ROSS, LIBERAL
GRIMES, JAMES T, LYONS
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GRDHS, HEINZ K, WICHITA
GROSSMAN, HARVEY M, SHAWNEE MISSION
GROVE, JOHN A, NEWTON
GROWNEY, JOHN T, ATCHISON
GRUENDEL, RICHARD A, KANSAS CITY
GRUENDEL, VIRGINIA T, KANSAS CITY
GRUND, FRANK M, WICHITA
GRUNOMEIEP, ANNETTE M, SHAWNEE MISSION
GRUSHNYS, ARNOLO, WICHITA
GSELL, GEORGE F, WICHITA
GUAROIA, OAVIO K, KANSAS CITY
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GUMUCIO, MARID L, KANSAS CITY
GUNN, MARVIN R, SALINA
GUNTER, CARL C, QUINTER
GUTHRIE, RICHARO A, WICHITA
GUTOVITZ, ALLEN LOUIS, TOPEKA
GUZMAN, MANUEL, SALINA
GWINN, OCUGLAS R, WICHITA

H

HA, SANG W, COFFEYVILLE
HABASHY, SHAWKY N F, WICHITA
HACKER, OAVIO CHARLES, SHAWNEE MISSION
HACKER, ELAINE MARY, TOPEKA
HAOLEY, OELMONT C, OTTAWA
HAFFNER, WILLIAM N, EL OORAOO
HAGAN, C THOMAS, WICHITA
HAGAN, FRANCIS J, WICHITA
HAGAN, ROBERT C, WICHITA
HAGGAN, MARGARET E, LAWRENCE
HAIGLER, JAMES P, HAYS
HAINES, ROBERT A, TOPEKA
HAIRE, WILLIAM O, HALSTEAO
HALE, RALPH, WICHITA
HALL, J ROGER, WICHITA
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HALLEY, M MARTIN, TOPEKA
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HALPIN, EOWARO O, WICHITA
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HAMILL, JOHN M, KANSAS CITY
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HAN, CHAN S, COFFEYVILLE
HANCOCK, ALAN C, KANSAS CITY
HANCOCK, DANIEL E, MANHATTAN
HANOLEY, OENNIS MICHAEL, SHAWNEE MISSION
HANOSHY, STANLEY E, ERIE
HANSA, HANSA, HAYS
HANSEN, FRANK W, GAROEN CITY
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HAQUE, ANWAR U L, IOWA CITY,IA
HARA, GLENN S, KANSAS CITY
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HARO, BENJAMIN F, KANSAS CITY,MO
HARDIN, CREIGHTON A, KANSAS CITY
HARMS, ALBERT C, SHAWNEE MISSION
HARMS, EDWIN M, WICHITA
HARMS, WILMER A, HALSTEAO
HARPER, OIANE M, KANSAS CITY
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HASTINGS, MARY T, SHAWNEE MISSION
HATHAWAY, PETER, SHAWNEE MISSION
HATTON, OONALO W, LAWRENCE
HATTON, LLOYO W, SALINA
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HAVENHILL II, MARSHALL A, EMPORIA
HAY, JAMES R, SHAWNEE MISSION
HAYES, WILLIAM L, WICHITA
*HAYNES, DEBORAH G, WICHITA
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HEBBAR, SATYA N, TOPEKA
HEORICK, KENNETH E, HUTCHINSON
HENOERSON, CHARLES F, PARSONS
HENOERSON, DAVID V, GARNETT
HENDRICKSON, JON R, NEWTON
HENORICKSON, KATHRYN D, NEWTON
HENNING, CALVIN W, OTTAWA
HENNING, CHARLES E, WICHITA
HENNING JR, HAROLD JOHN, KANSAS CITY
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HEREO, JOHN, WICHITA
HERMES, RICHARO L, LAWRENCE
HERMRECK, ARLO S, KANSAS CITY
HERRERA, JORGE J, TOPEKA
HERRMAN, AOAM L, SALINA
HERSHBERGER, GROVER, O.O., WICHITA
HERSHORN, SIMON E, WICHITA
HESSE, FREOERICK J, SALINA
HESSER, HERBERT H, SHAWNEE MISSION
HETTINGER, MICHAEL E, KANSAS CITY
HICKMAN, JAMES STEPHEN, TOPEKA
HIEBERT, ABRAHAM E, WICHITA
HIEBERT, OAVIO L, LAWRENCE
HIEBERT, JOHN B, TOPEKA
HIEBERT, JOHN M, KANSAS CITY
HIESTERMAN, HERMAN W, QUINTER
HILOYARO II, VICTOR H, COLBY
HILL, JAMES E, BELLA VISTA,AR
HILL, JOHN J, GREEN VLY,AZ
HILL, LARY MICHAEL, GREAT BENO
HILL, RICHARO H, MEADE
HILL, ROBERT N, TOPEKA
HILL, ROONEY W, SHAWNEE MISSION
HILST, WILBUR O, TOPEKA
HINSHAW, ALFREO H, WICHITA
HINSHAW, CHARLES T, WICHITA
HINSHAW, EOGAR D, ARKANSAS CITY
HINSHAW JR, CHARLES T, WICHITA
HINTHORN, OANIEL R, KANSAS CITY
HIRATZKA, TOMIHARU, WICHITA
HIRO, WAYNE E, LAWRENCE
HIRSCHBERG, J COTTER, TOPEKA
HISZCZYNSKYJ, ROMAN, TOPEKA
HITCHCOCK, C THOMAS, SHAWNEE MISSION
HIZON, RAMON R, WICHITA
HOAOLEY, WILLIAM O, KANSAS CITY
HOBBS, OONALO O, TOPEKA
HOBSON, MILBURN W, SHAWNEE MISSION
HOOES, HERBERT C, SHAWNEE MISSION
HODGES, BRUCE E, SHAWNEE MISSION
HOOGES, GLENN R, KANSAS CITY
HOOGES, MERLE A, SALINA
HOOGSON, OAVIO K, WASHINGTON
HODGSON, ROBERT B, WICHITA
HOOSON, OON W, MARION
HOOSON, HERVEY R, WICHITA
HOFER, OEWAYNE O, CONCORDIA
HOFFER, JOHN G, MEOICINE LODGE
HOHERZ, OAVIO G, TOPEKA
HOHLY, EVE K, KANSAS CITY,MO
HOLCOMB, OONALO G, LOS ANGELES,CA
HOLCOMB, WILLIAM M, LIBERAL
HOLOCRAFT, JACQUELYNE, KANSAS CITY
HOLOEN, OAVIO M, WICHITA
HOLOEN JR, RAYMOND F, WICHITA
HOLOERMAN, WALLACE O, HUTCHINSON
HOLLAOAY, HARMON M, WICHITA
HOLLAOAY, KENNETH R, EUOORA
HOLMAN, JON B, SALINA
HOLMES, FREOERICK F, KANSAS CITY
HOLMES, GRACE E, KANSAS CITY
HOLMES, JEO, WICHITA
HOLMES, JOHN A, KANSAS CITY
HOLMES, ROBERT W, TOPEKA
HOLSINGER, OONALO M, PITTSBURG
HOLT, JOHN M, GREAT BENO

HOOFER, WILFORO O, HALSTEAO
HOOPER, ALLEN, HOISINGTON
HOPKINS, WILLIAM O, SHAWNEE MISSION
HOPKINS JR, B MORRISON, SCOTT CITY
HOPPER, CHARLES R, EMPORIA
HORBELT, OOUGLAS V, WICHITA
HORNE, JAMES B, TOPEKA
HORSEMAN, ROBERT F, SHAWNEE MISSION
HORTON, WILLIAM A, KANSAS CITY
HOSTETTER, JAMES P, TOPEKA
HOSTETTER, PHILIP H, MANHATTAN
HOUSHOLOER, OANIEL FAIR, WICHITA
HOUSHOLOER, MARTHA S, WICHITA
HOUSTON II, LAWRENCE MORLEY, SHAWNEE MISSION
HOWARO, OONALO O, WICHITA
HOWELL, BARBARA JOYCE, EMPORIA
HOWERTER JR, BERNARO E, COFFEYVILLE
HOYLE, WINTHROP E, ESTES PARK,CO
HOYT, ARTHUR W, TOPEKA
HSU, CHENG H, TOPEKA
HSU, SHIN-FU, TOPEKA
HUAMAN, ANTONIO M, *,
HUANG, GEORGIANA L W, SHAWNEE MISSION
HUANG, JONSON, TOPEKA
HUOSON, ROBERT P, OLATHE
HUEBERT, OEAN A, WICHITA
HUEBNER, ROBERT STEPHAN, PITTSBURG
HUERTER, OAVID F, PITTSBURG
HUERTER, QUENTIN C, KANSAS CITY
HUGHES, ROBERT WALTER, LAWRENCE
HULL, KENNETH L, WICHITA
HULTGREN, MYRON K, WICHITA
HUME, JOSEPH W, WICHITA
HUMMER, LLOYO M, WICHITA
HUMPHREY, LOREN JENKINS, SHAWNEE MISSION
HUNO, LARRY R, WICHITA
HUNNINGHAKE, RONALO, SALINA
HUNSBERGER, TERRY R, O.O., GARDEN CITY
HUNTER, KENNETH R, LEBO
HUNTER JR, JAMES S, MANHATTAN
HURWITZ, ARYEH, KANSAS CITY
HUSEMAN, RICHARO ALLAN, NORTH KANS CITY,MO
HUSTEAO, ROBERT F, WICHITA
HUSTON, FRANCIS W, WINCHESTER
HUSTON, JOSEPH W, TOPEKA
HUTCHINSON, OIRK T, WINFIELO
HUTCHISON, GLEN C, HAYS
HUTCHISON, MARC K, HAYS
HUTTON, FREOERICK A, TOPEKA
HWA, EUGENE C, NEWTON
HYLAND, JOSEPH M, TOPEKA
HYMAN, HENRY T, O.O., KANSAS CITY,MO
HYNES, HARRY E, WICHITA

I

IBARRA, J LUIS, WICHITA
IBARRA, RICHARO C, KANSAS CITY
IOBEIS, BAOR, WICHITA
IHRIG, ROGER W, HUGOTON
ILIFF, R OOUGLAS, TOPEKA
ILIOPOULOS, JOHN I, KANSAS CITY
ILORETA, ALFREOO T, TOPEKA
INGHAM JR, H LAIRO, LAWRENCE
INGRAM, JOHN E, KANSAS CITY
INNES, ROBERT C, SHAWNEE MISSION
IRBY, AOOISON C, FORT SCOTT
IRBY, PRATT, FORT SCOTT
IRWIN, RICHARO L, NEWTON
ISAAC, CHARLES A, NEWTON
ISAACSON, RICHARO N, TOPEKA
ITURPALOE, GEORGE, SHAWNEE MISSION
IWAY, BELINO O, ELKHART
IWAY, OLIVIA N, ELKHART

J

JABEL, JUVENAL T, SATANTA
JACKMAN, GLENN H, ESTES PARK,CO
JACKS, J WARREN, HAWASEE,AZ
JACKSON, CATHERINE M, WICHITA
JACKSON, CHARLES R, WICHITA
JACKSON, LINOA H, TOPEKA
JACKSON, MICHAEL O, GAROEN CITY
JACKSON, ROBERT V, SHAWNEE MISSION
JACKSON, ROGER PAUL, N KANSAS CITY,MO
JACKSON, VICTOR L, ALTAMONT
JACKSON JR, OELMAS A, SALINA
JACOB, KANNAMPALLY L, EL OORAOO
JACOBS, OAVIO S, KANSAS CITY
JACOBS, RAE R, KANSAS CITY
JACOBSEN, OWIGHT SKINNER, COLBY
JACOBSON, JOEL A, WAKEENEY
JACOBY II, ROBERT E, TOPEKA
JAHANIAN, OARYOUSH, KANSAS CITY
JAMBOR, CHRISTOPHER N, OOOGE CITY
JAMES, OONALO L, WICHITA
JAMES, VERNON L, WICHITA
JANES, OONALO R, SHAWNEE MISSION
JANSSEN, ERWIN T, TOPEKA
JARROTT, JOHN B, HUTCHINSON
*JAWAOI, JAMEELA HUSAIN, WICHITA
JAYARAM, MARANOAPALLI R, KANSAS CITY
JAZAYERLI, NABIL, WICHITA

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JEHAN, SAYED S, WICHITA
JENNEY, CHARLES O, WICHITA
JENSEN, THOMAS M, OLATHE
JESTER, SHELBY L, WICHITA
JETER, JOHN HORTON, KANSAS CITY,MO
JEWELL, ROSS L, ST FRANCIS
JEWELL, WILLIAM R, KANSAS CITY
JIRICKO, MILOS, COFFEYVILLE
JOHLER, TERRY HARTWIG, LEAVENWORTH
JOHNS JR, LEO E, KANSAS CITY,MO
JOHNSON, BRUCE E, KANSAS CITY
JOHNSON, CAROL ANN, WICHITA
JOHNSON, CYNDA A, KANSAS CITY
JOHNSON, FREDERICK E, KANSAS CITY,MO
JOHNSON, GEORGE K, WICHITA
JOHNSON, HOWELL O, DODGE CITY
JOHNSON, J RICHARD, MCPHERSON
JOHNSON, JOHN E, KANSAS CITY
*JOHNSON, LEE ROY, TOPEKA
JOHNSON, PAUL O, LEAVENWORTH
JOHNSON, RICHARD L, HUTCHINSON
JOHNSON, THOMAS E, WICHITA
JONES, CHARLES E, SHAWNEE MISSION
JONES, EDWARD L, GREAT BEND
JONES, FORREST H, COLUMBUS
JONES, H IVOR, SHAWNEE MISSION
JONES, H PENFIELD, LAWRENCE
JONES JR, HERMAN H, KANSAS CITY
JOSEPH, BRIAN W, TOPEKA
JOSEPH, HOWARD F, LAWRENCE
*JOSEPH, JAPHET G, WICHITA
JOSS, CHARLES S, TOPEKA
*JOST, GARY O, WICHITA
JOUVENAT, NEIL C, SHAWNEE MISSION
JOYCE, G BERNARD, TOPEKA
JUBELT, HILBERT P, MANHATTAN
JUDILLA JR, FRANCISCO, WICHITA
JUSTUS, WILLIAM J, PLEASANTON

K

KADIAN, RAJESH S, SHAWNEE MISSION
KADISON, HERBERT I, WICHITA
KAGAN, STUART M, SHAWNEE MISSION
KALBAC, RICHARD W, GARDEN CITY
KALDOR, RICHARD H, MANHATTAN
KALIVAS, JAMES T, KANSAS CITY
KANE JR, WILLIAM M, HAYS
KANNEL, PHILIP D, KANSAS CITY
KARDATZKE, E STANLEY, WICHITA
KARDATZKE, JON K, WICHITA
KARLIN, CHARLES A, SAN DIEGO,CA
KARLIN, RONALD J, SHAWNEE MISSION
KARNAZE, MICHAEL G, KANSAS CITY
KASHA, ROBERT L, WICHITA
KASHYAP, BANSHI PRASAD, SHAWNEE MISSION
KASSEBAUM, GLEN E, EL DORADO
KASSEBAUM, KENNETH G, WICHITA
KAUFMAN, EUGENE E, WICHITA
KAUFMAN, LELAND R, WINFIELD
KAUFMAN, WILLARD E, MOUNDRIDGE
KAUL, ANAND N, WINFIELD
KAVEL, KARL K, TOPEKA
KAVI, NAGESH G, LEAVENWORTH
KEARNS, NORBERT W, TOPEKA
KEELER, LINDA L, KANSAS CITY
KEENE, GEORGE H, WICHITA
KEITH, ROBERT MARSHALL, MAPLE HILL
KELLER, JAMES P, WICHITA
KELLERMAN, RICK, PLAINVILLE
KELLEY, MARSHALL O, SHAWNEE MISSION
KELLING, COLLYER, EMPORIA
KELLY, A CHRISTINE, HAYS
KELLY, DAN A, TOPEKA
KELLY, ROBERT W, WICHITA
KENDALL, TOM E, WICHITA
KENDRICK, J GILLERAN, WICHITA
KENNALLY, KEVIN P, SABETHA
KENNEDY, GERALD T, WICHITA
KENNEDY, JAMES A, KANSAS CITY
KENNEDY, KENNETH R, SHAWNEE MISSION
KEPES, JOHN J, KANSAS CITY
KEPKA MD, DENNIS J, HUTCHINSON
KERBY, GERALD R, KANSAS CITY
KESTENBAUM, THELMA M, KANSAS CITY
KETCHERSIDE JR, WILLIAM J, BLUE SPRINGS,MO
KETCHUM, LYNN O, SHAWNEE MISSION
KEYS JR, ROBERT C, TOPEKA
KEYS SR, ROBERT C, TOPEKA
KHICHA, GYANCHAND J, WICHITA
KHOURY, GEORGE H, WICHITA
KHURANA, SATISH K, SHAWNEE MISSION
KIFER, C JAMES, HAYS
KIHM, ALBERT A, CHANUTE
KIM, JONG M, KANSAS CITY
KIM, PAIK N, WICHITA
KIM, YONG W, TOPEKA
KIMBALL, RICHARD R, MANKATO
KIMBLE, JAMES A, WICHITA
KIMMEL, KENNETH K, HALSTEAD
KIMURA, CHARLES C, SHAWNEE MISSION
KINDLING, PAUL H, TOPEKA
KING, CHARLES R, KANSAS CITY
KING, TERESA M, KANSAS CITY
KING, WILLIAM T, GREAT BEND

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KINPORTS SR, EDWARD B, KANSAS CITY,MO
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KIRCHNER, FERNANDO R, KANSAS CITY
KIRK, THOMAS E, MANHATTAN
KIRK JR, E DAVID, WICHITA
KIRKEGAARD, ROGER S, TOPEKA
KISER, JOHN L, WICHITA
KISER, WILLARD J, WICHITA
KISHORE, ROY N, PARSONS
KISHORE, SHEELA, PARSONS
KITCHEN, ROBERT R, WICHITA
KLEINHOLZ JR, EMIL JOHN, TOPEKA
KLEMMER, HERBERT, TOPEKA
KLENDA JR, MARTIN B, BELOIT
KLIEWER, VERNON L, NEWTON
KLINGLER JR, EUGENE A, MANHATTAN
*KLINGMAN, DIANE D, ANDOVER
KLOBASA, CHARLES L, MANHATTAN
KLONTZ, WILLIAM JOSEPH, WICHITA
KLOSTERHOFF, BRUCE E, HUTCHINSON
KNAPP, LESLIE E, WICHITA
KNAPP, M ROBERT, WICHITA
KNAPPENBERGER, ROY C, MANITOU SPRING,CO
KNECHT, STEPHEN M, EMPORIA
KNEIDEL, THOMAS W, WICHITA
*KNIGHT, LAURA C, WICHITA
*KNIGHT, PHILIP J, WICHITA
KNUTH, KENNETH L, INDEPENDENCE
KODANAZ, A AYTEKIN, SHAWNEE MISSION
KOKSAL, TOM, GARDEN CITY
KOONS, JESS W, LIBERAL
*KOONTZ, JUDITH A, TOPEKA
KOSAR, CLARENCE O, CONCORDIA
KOURI, SAMMY H, WICHITA
KOVAC, ANTHONY L, KANSAS CITY
KOVARIK, ERNEST O, TOPEKA
KOZIKOWSKI, BEN M, SHAWNEE MISSION
KRANTZ, KERMIT E, KANSAS CITY
KRAUSE, ROLAND L, WICHITA
*KREADY, JOHN L, WICHITA
KREHBIEL, MARK A, SALINA
KRETSINGER DD, W BROCK, EMPORIA
KROLL, HARRY G, TOPEKA
KRUCKEMYER, ALAN L, SALINA
KRUEGER, HAVEN C, GREAT BEND
KRUEGER, KURT ALLEN, KANSAS CITY,MO
KRUPKA, JOHN J, WICHITA
KUBIN, DORIS A, SHAWNEE MISSION
KUBINA, GLENN RICHARD, WICHITA
KUEBLER, KEVIN M, KANSAS CITY
KUMAR, SURINDER, NEWTON
KURTH, C JOSEPH, WICHITA
KURTH, PAUL H, SHAWNEE MISSION
KURTH, ROBERT H, SHAWNEE MISSION
KUTILEK, FRANK J, WICHITA
KWEE, SIOE T, KANSAS CITY
KYNER, JOSEPH L, KANSAS CITY

L

LABHSETWAR, S A, JUNCTION CITY
LACCHEO, MICHAEL L, TOPEKA
*LACEY, STEFFAN R, WICHITA
LAFENE, BENJAMIN W, MANHATTAN
LAHAM, ALEXANDER J, DALLAS,TX
LAI, CHI-WAN, KANSAS CITY
LAI, JENG Y, WICHITA
LAI, MAX G, TOPEKA
LAI-CHEN, SHEUE-HUEY, TOPEKA
LAING, ROBERT R, KANSAS CITY
LAIRD, DALE D, OLATHE
LAKE, MAX S, SALINA
LANCE, RAYMOND W, PITTSBURG
LANCE JR, JOHN F, WICHITA
LAPI, RUTH M, SHAWNEE MISSION
LARSON, DELBERT L, HIAWATHA
LASH, RAY E, SHAWNEE MISSION
LASLEY, DAVID A, SALINA
LASLEY, MICHAEL B, HAYS
LATIMER, KATHERINE, WICHITA
LAUNEY, WALTON S, TOPEKA
LAURY, DAVID G, OTTAWA
LAUVER, MARY ANN, WICHITA
LAVA, CHIRUNO, PARSONS
LAW, FINDLEY, ELLINWOOD
LAWHORN, STEPHANIE LU, KANSAS CITY
LAWLESS, HAROLD L, BLUE RAPIDS
LAWN, CLAUDIA A, WICHITA
LAWN, RAYMOND A, WICHITA
LAWRY, JAMES MORIS, WINFIELD
LAWS, LEWIS R, MARYSVILLE
LAWSON, DWIGHT, TOPEKA
LAWTON, MARVIN K, CONCORDIA
LAWWILL, THEODORE, KANSAS CITY
LAYBOURNE JR, PAUL C, KANSAS CITY
LAZAR, HARRY, WICHITA
LEARNED, GEORGE R, LAWRENCE
LEATHERS III, HOLLIS K, SHAWNEE MISSION
LEE, CATHERINE, TOPEKA
LEE, JAE M, KANSAS CITY
LEE, KYO R, KANSAS CITY
LEE, R REX, WICHITA
LEE, SANG DUG, FT DODGE,IA
LEE, SONG DOW, TOPEKA
LEE, SONG PING, TOPEKA
LEE, YONG U, EL DORADO
LEE JR, EDWARD S, WICHITA
LEE JR, JAMES G, KANSAS CITY
*LEFEVRE, JOHN R, D.D., TOPEKA
LEFFLER, PAUL B, PITTSBURG
LEGASPI JR, PEDRO L, SHAWNEE MISSION
LEGER, LEE H, FT MYERS,FL
LEGLER, GARY LEE, D.D., TOPEKA
LEIFER, WILLIAM N, TOPEKA
LEIKER, JOSEPH, ARKANSAS CITY
LEISY, JERALD W, WICHITA
LEITCH, DAVID A, GARNETT
*LEITNER, YORAM B, WICHITA
LEMOINE JR, ALBERT N, KANSAS CITY
LENEVE, ROBERT T, HUGOTON
LENSKI JR, FRANCIS X, IOLA
LENTZ, WILLIAM R, TOPEKA
LEO, WILLIAM A, KANSAS CITY
LERNER, SCOTT A, SHAWNEE MISSION
LESSENDEN, GLENN A, LAWRENCE
LESSENDEN JR, C M, TOPEKA
LESSER, DANE A, HUTCHINSON
LESTER, JOHN BUCKLES, SHAWNEE MISSION
LETTNER, HANS T, HUTCHINSON
LEVINE, ERROL, KANSAS CITY
LEVINE, WILLIAM R, WICHITA
LEVY, EDWIN Z, TOPEKA
LEWIN, WALTER, SHAWNEE MISSION
LEWIS, JAMES E, SHAWNEE MISSION
LEWIS, LARRY D, WICHITA
LEWIS JR, H DANIEL, KANSAS CITY,MO
LIEBERMAN, BRUCE IRWIN, KANSAS CITY
LIES, BARTHEL N, COLWICH
LIES, RICHARD B, WICHITA
LIESMANN, GEORGE E, TOPEKA
LIESMANN, JEAN ELIZABETH, TOPEKA
LIN, JOE J, WICHITA
LINO II, EDWARD J, GARDEN PLAIN
LINDHOLM, GERALD R, NEWTON
LINOSLEY, CAROL B, KANSAS CITY
LINOSLEY, HERBERT B, KANSAS CITY
LINHARDT, RONALD D, WICHITA
LINN, CATHERINE P, KANSAS CITY
LINSHAW, MICHAEL A, KANSAS CITY
LIPOFF, JAY I, LEAVENWORTH
LIPSEY, JAMES H, SHAWNEE MISSION
LITTELL, JAMES A, WICHITA
LITTLE, L GILBERT, WICHITA
LIU, ALBERT T, KANSAS CITY
LIU, CHIEN, KANSAS CITY
LIVINGSTON, CHARLES E, SALINA
LLOYD, HARVEY L, KANSAS CITY
LLOYD, JAMES W, CONCORDIA
LOCKHART, JOSEPH G, WICHITA
LOEB, ELBIE L, HAYS
LOEFFLER, JAMES A, WICHITA
LOEWEN, HENRY H, WICHITA
LOEWEN, PETER S, HILLSBORO
LOEWEN, WILLIAM C, WICHITA
LOGAN, GEOFFREY G, WICHITA
LOGANBILL, VARDEN J, MOUNDRIDGE
LOHMEYER, KENNETH L, GREEN VALLEY,AZ
LOHRENTZ, LOIS H, SHAWNEE MISSION
LONEY, JOHN M, BELOIT
LONG, EDWARD E, HUMBOLDT
LONG, JOHN W, TOPEKA
LONG, LLOYD O, GOODLAND
LONG, ROBERT C, NORTON
*LOSEE, JOHN M, WICHITA
LOTITO, CARLOS A, KANSAS CITY
LOVELAND, G CHARLES, LAWRENCE
LOVETT, PAUL A, WICHITA
LOW, HAROLD L, WICHITA
LOWE, STANLEY W, MANHATTAN
LOWMAN, JAMES T, KANSAS CITY
LUEGER, JAMES JOHN, D.D., EMPORIA
LUEGER DD, JAMES JOHN, SENECA
LUEKEN, LUEKE B, WICHITA
LUELLEN, THOMAS J, WICHITA
LUETJE, CHARLES MARION, KANSAS CITY,MO
LUKENS, DAVID, HUTCHINSON
LUKERT, BARBARA P, KANSAS CITY
LULO, ANTONIO R, SHAWNEE MISSION
LUMB, RAYMOND C, TOPEKA
LUNGSTRUM, JACK E, SALINA
LUZZATI, ENZO F, WICHITA
LYNCH, JOHN A, TOPEKA
LYNCH, MARY A, WICHITA
LYNCH, SEAN R, KANSAS CITY
LYONS JR, FRANK C, MANHATTAN

M

MAC KILLOP JR, DANIEL, WINFIELD
MACARTHUR, RICHARD IAN, KANSAS CITY
MACDOUGALL, MARGARET L, SHAWNEE MISSION
MACE, RONALD D, JUNCTION CITY
MACY, NORMAN E, SALINA
MACY, TED L, SALINA
MADISON, WILLARD A, NORTONVILLE
MADISON JR, WARD N, WICHITA
MADSEN, GLENN L, LAWRENCE
MAGEE, LAWRENCE M, WASHINGTON
*MAGNUSON, ELLIOT ARTHUR, WICHITA
MAGRINA, JAVIER F, KANSAS CITY
MAILMAN, GERSHOM, WICHITA
MALLORY, JOHN A, SHAWNEE MISSION
MALONE, EUGENE M, HALSTEAD

MANAHAN, G EUGENE, LAWRENCE
MANGOLD, JOEL VOYCE, KANSAS CITY
MANI, MANI M, KANSAS CITY
MANLEY, JOSEPH W, SHAWNEE MISSION
MANNING, ROBERT T, WICHITA
MANSFIELD, CARL M, KANSAS CITY
MANSOUR, BADIE S, WICHITA
MANTZ, FRANK A, SHAWNEE MISSION
MARCHBANKS, DONALD L, SALINA
MARPLES, BRADLEY W, KANSAS CITY
MARR, GARRETT L, SHAWNEE MISSION
MARSH, ALICE GARRISON, KANSAS CITY
MARSH, CONNIE M, HALSTEAD
MARSH, HENRY O, WICHITA
MARSHALL, B M, TOPEKA
MARSHALL, GEORGE D, BELLA VISTA AR
MARSHALL, GEORGE W, SALINA
MARSHALL, RONALD L, MANHATTAN
MARTIN, DANIEL C, MANHATTAN
MARTIN, EARL A, PARSONS
MARTIN, JOSEPH P, KANSAS CITY
MARTIN, NORMAN L, KANSAS CITY
MARTIN, OLIVER L, SALINA
MARTIN, RONALD L, KANSAS CITY
MARTIN, WILLIAM O, TOPEKA
MARTIN JR, GLEN E, WICHITA
MARTINEZ, JOHN D, HUTCHINSON
MARTINEZ, KAY C, PEABODY
MARVEL, JAMES EBBERT, ARKANSAS CITY
MARVIN, NORMAN G, SHAWNEE MISSION
MARYMONT JR, JESSE H, WICHITA
MASER, GEORGE R, SHAWNEE MISSION
MASSEY, ANDREW O, WICHITA
MASTERS, FRANCIS W, KANSAS CITY
MASTERSON, MELVIN LEROY, TROY
MASTIO JR, GEORGE J, WICHITA
MATASSARIN, BENJAMIN M, WICHITA
MATASSARIN, FREDERICK W, WICHITA
MATHEWS, MICHAEL WM, OLATHE
MATHEWS, ROBERT C, OLATHE
MATHEWS, ROBERT MAJOR, SHAWNEE MISSION
MATHEWSON, HUGH S, KANSAS CITY
MATHIS, JERRY L, SALINA
MATTA, JOSE R, DODGE CITY
MATTHEW, WILLIAM L, OLATHE
MATTICK, IRVIN H, HAYS
MATTIOLI, LEONE, KANSAS CITY
MAU, WALTER, TOPEKA
MAUCK, HAROLD C, STOCKTON
MAWDSLEY, MICHAEL W, WICHITA
MAXFIELD, RUSSELL J, COLORADO SPRING,CO
MAXWELL, GORDON E, SALINA
MAXWELL, ROBERT A, SHAWNEE MISSION
MAY, KENNETH L, BONNER SPRINGS
MC GEENEY, TERRY L, SENECA
MC NICKLE, GEORGE ANDREW, WICHITA
MC PHEE, MARK S, KANSAS CITY
MCALLASTER, WENDALE E, GREAT BEND
*MCBDYLE, MARILEE, WICHITA
MCCANN, PATRICK E, FORT SCOTT
MCCANN, WILLIAM E, OLATHE
MCCARTER, DUANE K, TOPEKA
MCCARTHY, ROBERT P, KANSAS CITY
MCCAUGHEY, HUGH W, SHAWNEE MISSION
MCCLANAHAN, WARD A, WICHITA
MCCLELLAN, ERNEST L, WICHITA
MCCLELLAN, JOHN W, TOPEKA
MCCLURE, JAMES A, TOPEKA
MCCOLLUM, WILLIAM O, LEAVENWORTH
MCCOMAS JR, MARMADUKE D, TOPEKA
MCCOWEN, HERBERT M, SHAWNEE MISSION
MCCOY, CHARLES P, WICHITA
MCCOY, CHARLES T, HUTCHINSON
MCCOY, MICHAEL T, TOPEKA
MCCOY, RONALD, DODGE CITY
MCCRAE, SPENCER C, SALINA
MCCULLOUGH, ROBERT C, GOODLAND
MCCUNE, MARK A, KANSAS CITY
*MCDONOUGH, WILLIAM O, WICHITA
MCEACHEN, WILLIAM H, SHAWNEE MISSION
MCELHINNEY, CHARLES F, DODGE CITY
MCELROY, MICHAEL B, SHAWNEE MISSION
MCELROY, ROBERT T, TOPEKA
MCELROY, WILBUR J, TOPEKA
MCGINNIS, MICHAEL J, DODGE CITY
MCGUIRE, WILLIAM F, WICHITA
MCGURK, THOMAS E, SHAWNEE MISSION
MCKEE, LEO F, COTTONWOOD FALS
MCKENNA, MICHAEL J, FORT SCOTT
MCKERPACHER, ROBERT D, DERBY
MCKIM, W LYNN, KINSLEY
MCKNIGHT, DAVID E, MANHATTAN
MCKNIGHT, ELLIS B, ALMA
MCLAIN, KENNETH, RANSOM
*MCMULLEN, BRUCE R, WICHITA
MCMULLEN, JOSEPH E, HUTCHINSON
MCNEIL, ELBERT O, MANHATTAN
*MCQUEEN, DAVID ARNOLD, WICHITA
MCVAY, R BRUCE, CLAY CENTER
MCVEY, PAMELA S, KANSAS CITY,MO
MCWHERTER, LOTTIE B, SHAWNEE MISSION
MEADOWS, DONALD C, WICHITA
MEBUST, WINSTON K, KANSAS CITY
MECH, ARNOLD W, TOPEKA
MEDHAT, MOHAMED A, KANSAS CITY
MEE, ADRIAN W, OLATHE
MEEK, PALMER F, MANHATTAN
MEEK JR, JOSEPH C, KANSAS CITY
MEEKER II, BRUCE P, WICHITA

* Probationary members.

MEGIBOW, ALAN D, TOPEKA
MEHAFFY, ORVILLE A, JOPLIN,MO
MEIDINGER, RAY, HIAWATHA
MEIDINGER, RICHARD, TOPEKA
MELEAN, JAIME, WICHITA
MELENDRES, JUANITO M, OLATHE
MELHORN, J MARK, WICHITA
MELHORN MD, KATHERINE J, WICHITA
MELIN, BRUCE O, GARDEN CITY
MENAKER, JEROME S, WICHITA
MENDIONES, L MARLENE, WICHITA
MENDIONES, RUPERTO D, WICHITA
MENOLICK, R MICHAEL, OLATHE
MENEHAN, H JAMES, WICHITA
MENEZ, CESAR V, SHAWNEE MISSION
*MENHUSEN, MONTY J, D.O., WICHITA
MENKING, F W MANFRED, WICHITA
MENKING, SUSAN MARGARET, WICHITA
MENNINGER, KARL A, TOPEKA
MENNINGER, ROBERT G, TOPEKA
MENNINGER, ROY W, TOPEKA
MENNINGER, W WALTER, TOPEKA
MERCADER, MARIO S, WICHITA
MEREDITH, W TOM, WICHITA
MERKEL, EARL D, RUSSELL
MERRITT, JOE P, WICHITA
MERRITT, W HENRY, LEAVENWORTH
MERSHON, JAMES C, WICHITA
MESINA, ROLANDO R, KANSAS CITY
MESROPIAN, GEORGE O, ATCHISON
MEYER, MARK E, SHAWNEE MISSION
MEYER, O WARREN, TOPEKA
MEYER, WARREN E, WICHITA
MEYERS, STEPHEN, GARDEN CITY
MICHELBACH, ALBERT P, WICHITA
MIGUELINO, OLIVER M, EMPORIA
MIH, ALEXANDER, CHANUTE
MILFELD, DOUGLAS J, WICHITA
MILLER, ABRAHAM H, MANHATTAN
MILLER, CHARLES H, PARSONS
MILLER, CLYDE W, WICHITA
MILLER, DAVID PATERSON, WICHITA
MILLER, DEAN M, PARSONS
MILLER, DON E, WICHITA
MILLER, EARL E, PITTSBURG
MILLER, ELDEN V, SALINA
MILLER, FRANKLIN R, WINFIELD
MILLER, FREEMAN LANCE, SHAWNEE MISSION
MILLER, HERBERT C, NORFORD,CT
MILLER, LAWRENCE H, DERBY
MILLER, MONTE B, SCOTT AFB,IL
MILLER, PHILIP A, PHOENIX,AZ
MILLER, ROBERT E, GARDEN CITY
MILLER, STEPHEN FRANCIS, PARSONS
MILLIGAN, DONALD B, KANSAS CITY
MILLS, CHARLES D, WICHITA
MILLS, GEORGE QUINTON, WICHITA
MILLS, VERNON A, LEAVENWORTH
MILLS JR, PHILIP E, TOPEKA
MIMOSO, JOSE J, DODGE CITY
MINER JR, PHILIP B, KANSAS CITY
MINNICK, CHARLES V, JUNCTION CITY
MINNS, GAROLD O, WICHITA
MIRZA, MEOO, WICHITA
MISKEW, DON B W, SHAWNEE MISSION
MITCHELL, ALEX C, LAWRENCE
MITCHELL, JOHN C, SALINA
MITTS, ERNEST W, BONNER SPRINGS
MODDRELL, CAROL A, LAWRENCE
MODLIN, HERBERT C, TOPEKA
MOELLER, DONALD D, KANSAS CITY
MOFFAT, ROBERT E, SHAWNEE MISSION
MOHLER, JACK M, ABILENE
MOHR, SANDRA NELL, WICHITA
MONCKTON, LAURANCE A, LAWRENCE
MONTGOMERY, LLOYD DAN, HALSTEAD
MONTGOMERY, THOMAS ALLEN, SARETHA
MONTGOMERYSHOPT, RUTH G, HALSTEAD
MOORE, DENNIS F, WICHITA
MOORE, HUGH C, TOPEKA
MOORE, JAMES E, CONCORDIA
MOORE, JANE A, WICHITA
MOORE, ROBERT, HOISINGTON
MOORE, ROBERT F, CANEY
MOORE, WAYNE V, KANSAS CITY
MOORHEAD JR, F ALLEN, NEODESHA
MORFFI, RAUL R, KANSAS CITY
MORGAN, DICK A, EAST BLUE HILL,ME
MORGAN, JAMES I, WICHITA
MORGAN, JOHN L, EMPORIA
MORGAN, NOVA L, HAYSVILLE
MORGAN II, DAVID LLOYD, OLATHE
MORGAN III, LOUIS S, WICHITA
MORITZ, RICK S, SHAWNEE MISSION
MORONEY, JEAN M, SHAWNEE MISSION
MORRIS, MERLE D, TOPEKA
MORRISON, IRA R, ATCHISON
MORRISON, MICHAEL R, TOPEKA
MORRISON, RICHARD L, WICHITA
MORROW, THOMAS F, WICHITA
MORROW JR, J TARLTON, TOPEKA
MORTON, JOHN E, HALSTEAD
MOSER, ERNEST C, HOLTON
MOSER, M ROSS, HOLTON
MOSER, ROY H, HOLTON
MOSIER, MICHAEL L, MANHATTAN
MOSIER, STANLEY JAY, WICHITA
MOSIER, STEVEN J, MANHATTAN
MOWERY, WILLIAM E, SALINA
MOWRY, GERALD L, MANHATTAN

MOYER, HERMAN J, DERBY
MUEHLBERGER, JAMES J, SHAWNEE MISSION
MUELLER, ARNOLD V, TOPEKA
MUELLER, J KENT, SHAWNEE MISSION
MUELLER, VERNETTE A, WICHITA
*MUETH, JOAN D, WICHITA
MULL, JOHN C, HUTCHINSON
MULLEN SR, CLIFFORD J, KANSAS CITY
MULLER, SAMUEL B, PITTSBURG
MULLINIX, JANICE M, WICHITA
MURFITT, MALCOLM C, LINDSBORG
MURPHY, BARRY L, WICHITA
MURPHY, DUANE A, WICHITA
MURPHY, JAY W, SHAWNEE MISSION
MURPHY, PAUL M, WICHITA
MURPHY, PAUL W, WICHITA
MURPHY, WILLIAM R C, WICHITA
MURRAY, KENT B, WICHITA
MURRAY, W LEE, SHAWNEE MISSION
MYERS, JO ANN, TOPEKA
MYERS, W EUGENE, IOLA
MYERS JR, EARL B, INDEPENDENCE
MYRICK, MICKEY, HAYS
MYRICK, STEPHEN W, LAWRENCE

N

NABOURS, RICHARD D, TOPEKA
NACHTIGALL, ANDREW, NEWTON
NALDOZA JR, FAUSTINO M, WELLINGTON
NANCE, JOEL, TOPEKA
NARAWONG, DUANGCHAI, WINFIELD
NARCISO, VICENTE D, ABILENE
NASH, NEWMAN CURTIS, SCOTTSDALE,AZ
NASH, ROBERT A, SHAWNEE MISSION
NAUER, PAULA LOU, SHAWNEE MISSION
NAVICKAS, LEONARD A, SHAWNEE MISSION
NEEL, WILBUR B, HUTCHINSON
NEFF, JAMES R, KANSAS CITY
NEIBURGER, JAMES B, SHAWNEE MISSION
NEIGHBOR, ERNEST G, KANSAS CITY
NEIGHBOR, ERNEST H, SHAWNEE MISSION
NEIGHBOR, GAYLORD P, KANSAS CITY
NEIGHBOR, RALPH M, EMPORIA
NEIL, ROY NEWTON, HAYS
NEIS, HARRY D, OSAGE BEACH,MO
*NELLIS, STEPHANIE F, WICHITA
NELSON, BRYAN C, SHAWNEE MISSION
NELSON, GERALD D, WICHITA
NELSON, JOHN B, SHAWNEE MISSION
NELSON, PAUL L, CONCORDIA
NELSON, RICHARD O, LAWRENCE
NELSON, RUSSELL ALAN, WICHITA
NELSON, T EUGENE, FORT SCOTT
NELSON, WILLIAM PAUL, KANSAS CITY
NELSON JR, GUST H, WICHITA
NEMMERS, DAVID J, WINFIELD
NESMITH, LESLIE W, WICHITA
NEUENSCHWANDER, JOHN, HOXIE
NEUENSCHWANDER, JOHN RAND, HOXIE
NEUER, FREDERICK S, EMPORIA
NEUSCHAFER, DARREL R, HUTCHINSON
NEVINS, RICHARD L, LIBERAL
NEVITT, J RUSSELL, MORAN
NEWBY, JAMES P, WICHITA
NEWCOMB, WARD M, HAYS
NEWKIRK, DARREL O, KANSAS CITY
NEWMAN, CARL T, CONCORDIA
NEWMAN, CLIFFORD B, PITTSBURG
NEWSOM, F CARTER, WICHITA
NEWTON, CHARLES R, VISALIA,CA
NIBBELINK, LARRY WAYNE, KANSAS CITY
NICE, G WILLIAM, TOPEKA
NICHOLS, JEFF S, TOPEKA
NICHOLS, RICHARD, COFFEYVILLE
NICHOLS, ROBERT R, FORT SCOTT
NICKELL, WENDELL K, SALINA
NIEDEREE, W CURTIS, GREAT BEND
NIELSEN, MARY L, HAYS
NIEMAN, JOHN L, SHAWNEE MISSION
NIENSTEDT, JOHN F, SUN CITY,AZ
NIKNIA, SEYED M, GARDNER
NIXON, JAMES F, DODGE CITY
NIXON, RICHARD R, SALINA
NIXON, WILLIAM A, WICHITA
NOBLE, MARK J, KANSAS CITY
NOHE, PHILIP C, KANSAS CITY
NORRIS, CHARLEY W, KANSAS CITY
NORRIS, ROBERT P, WICHITA
NORTH, DORIS G, WICHITA
NORTH, VICTOR, WICHITA
NORTHWAY, DANIEL P, TOPEKA
NORTON, ROBERT K, WICHITA
NOSTI, JUAN C, SHAWNEE MISSION
NOTHNAGEL, ARNOLD F, KANSAS CITY
NOTTINGHAM, ROBERT M, OLATHE
NOVOTNY, PETER C, TOPEKA
NOWLIN, NANCY S, WICHITA
NUILA, RICHARD F, WICHITA
NULL, WILLIAM G, SALINA
NUNEMAKER, MARION E, HUTCHINSON
NUNEZ, JULIAN, KANSAS CITY
NYBERG, FREDRIK F, TOWANDA
NYE, C ERIK, SHAWNEE MISSION

O

O'BOYNICK II, PAUL LEONARD, KANSAS CITY
O'BRYAN, JAMES J, SHAWNEE MISSION
O'CONNELL, FRANK A, SHAWNEE MISSION
O'DONNELL, HAROLD F, ELLSWORTH
O'DONNELL, HARRY E, JUNCTION CITY
O'DONNELL, RICHARD H, CLAY CENTER
O'DONNELL JR, LEONARD A, WICHITA
O'DONNELL SR, LEONARD A, WICHITA
O'NEIL, ROBERT H, TOPEKA
O'SHEA, JAMES G, JETMORE
O'TOOLE, JAMES K, NEWTON
OBANDO, GUILLERMO, SALINA
OBBURN, ROBERT L, TOPEKA
OCHSNER, BRUCE B, WICHITA
ODENHEIMER, BURTRAM J, WICHITA
ODGERS, RODNEY K, PITTSBURG
OELSCHLAGER, RONALD O, LAWRENCE
OHMAN, RICHARD J, DODGE CITY
OHMART, RICHARD V, OAKLEY
OKTAWIEC, DANUTA, SHAWNEE MISSION
OLD, JERRY L, ARKANSAS CITY
OLDFIELD, RAY W, KANSAS CITY
OLNEY, ROBERT O, MANHATTAN
OLSEN, PHILLIP S, EL DORADO
OLSON, CARL E, LAWRENCE
OLSON, CLITUS W, GOODLAND
OLSON, ERWIN T, NEWTON
OPENSHAW, CALVIN R, HUTCHINSON
ORCHARD, RICHARD A, LAWRENCE
ORR, STEVEN M, KANSAS CITY, MO
OSBERN, LIDA, LAWRENCE
OSBORN, ROBERT M, D.O., INDEPENDENCE
OSBORNE, CONRAD C, WICHITA
OSIO, ANTONIO L, WICHITA
OSOBA, WILLIAM G, WICHITA
OTHMER, EKKEHARD, KANSAS CITY
OUANO JR, BIBIANO B, WICHITA
OVERFIELD, A SCOTT, KANSAS CITY
OVERHOLSER, NORMAN H, EL DORADO
OWEN, GARRY O, LAWRENCE
OWEN, LARUE W, WICHITA
OWEN, PERE A, WICHITA
OWENS, RICHARD L, SHAWNEE MISSION
OWENSBY, L C, CONCORDIA
OXLER JR, JOHN EDWARD, KANSAS CITY

P

PACE, JOHN O, PARSONS
PADGETT, WILLIAM L, WICHITA
PAGE, O VALE, PLAINVILLE
PAGE, RUTH, WICHITA
PAI, RADHA V, PARSONS
PAI, VARADARAJ S, PARSONS
PAINE, GEORGE E, HUTCHINSON
PALMBERG, KENT E, TOPEKA
PALMER, DAVID L, WICHITA
PALMER, GERALD K, SALINA
PALMER, MARGUERITE L, OBERLIN
PALMER, MARVIN M, KANSAS CITY
PALMERI, MARIA, KANSAS CITY
PALMERI, V CARLOS, KANSAS CITY
PANICHABHONGSE, SAMBHUNDH, RUSSELL
PAPP JR, S DEAN, PITTSBURG
PARAMESH, JAYA, LARNED
PARANJOTHI, SUBRAMONIAM P, PARSONS
PARDO, MANUEL P, KANSAS CITY
PAREKH, AJITKUMAR M, KANSAS CITY
PARHAM, VERDON W, CHANUTE
PARK, CHAN H, KANSAS CITY
PARK, ROGER WALTER, WICHITA
PARKER, HAROLD L, WICHITA
PARKER, ROBERT W, LEAVENWORTH
PARKER, WAYNE G, EMPORIA
PARMAN, ROBERT D, TOPEKA
PARRA, MIGUEL D, KANSAS CITY
PARSI, MANUTCHEHR K, PITTSBURG
PARULKAR, DEEPAK S, TOPEKA
PASCHINO, ANTONIO R, KANSAS CITY
PASIMIO, ROGER S, COLUMBUS
PASTOR, VICTOR HUGO, EMPORIA
PATEL, MAHENDRA N, JUNCTION CITY
PATEL, V C, INDEPENDENCE
PATEL, VINOD, TOPEKA
PATINO, EDGAR, TOPEKA
PATRICK, FRED EDWARD, TOPEKA
PATTERSON, BRUCE W, WICHITA
PATTERSON, JOHN R, SHAWNEE MISSION
PATTON, J MICHAEL, WICHITA
PAULS, DANIEL N, PARSONS
PAY, NORMAN T, WICHITA
PAYNE, J RALPH, KANSAS CITY, MO
PAYNE, ROBERT R, TOPEKA
PAZELL, JOHN A, KANSAS CITY
PEARCE, EUGENE W J, SHAWNEE MISSION
PEARCE, LUNETTA M, SHAWNEE MISSION
PEASE, GARY L, HUTCHINSON
PECANA, MANUEL C, KANSAS CITY
PEDERSON, ARNOLD M, PLAINVILLE
PEDRAZA, HERNANDO, WELLINGTON
PEERY, WILLIAM H, WICHITA
PEES, GERALD B, IOLA
PEES JR, GERALD BOYD, LAWRENCE
PEFFLY, ELMER O, CHETOPA
*PELLETIER JR, LAWRENCE L, WICHITA
PENCE, CHARLES D, WICHITA
PENN, GEORGE M, TOPEKA
PENNER, TIMOTHY M, KANSAS CITY
PENNINGTON, KATHERINE, WICHITA
PENTECOST, RICHARD L, SHAWNEE MISSION
PERDUE II, W LANG, TOPEKA
PEREIRA, WILLY G, ARKANSAS CITY
PERICO, CARLOS J, KANSAS CITY, MO
PERIOO, DOMINADOR T, ELKHART
PERKINS, JACK L, HUTCHINSON
PERRY JR, LAWRENCE L, KANSAS CITY
PETELIN, JOSEPH B, SHAWNEE MISSION
PETERIE, JERRY P, WICHITA
PETERS, THOMAS J, WICHITA
PETERSEN, A GENE, SHAWNEE MISSION
PETERSEN, GERALD D, SHAWNEE MISSION
PETERSON, DEAN L, TOPEKA
PETERSON, JACK T, MANHATTAN
PETERSON, JAMES E, SALINA
PETERSON, ROBERT L, TOPEKA
PETERSON, VERNON J, TOPEKA
PETERSON JR, EVAN A, WATHENA
PETIT, CARL ALFONSO, SHAWNEE MISSION
PETRIE, SAMUEL C, SHAWNEE MISSION
PETRIK, EDWIN L, TOPEKA
PETTERSON, CECIL E, SYRACUSE
PETTERSON, DENNIS CRAIG, TOPEKA
PETTERSON, O'RUTH S, RIOGEVILLE, IN
PETTIJOHN, WALTER J, RUSSELL
PFUETZE, BRUCE L, SHAWNEE MISSION
PFUETZE, ROBERT E, TOPEKA
*PHAN, DUNG MY, WICHITA
PHELPS, DAVID WAYNE, FORT SCOTT
PHILGREEN, ODNALD E, OTTAWA
PHILIPP, JOSEPH THEODORE, MANHATTAN
PHILLIPS, STEPHEN B, MANHATTAN
PHILLIPS, WARREN G, SHAWNEE MISSION
PHIPPS, JACK G, WICHITA
PHIPPS, RONNY G, INDEPENDENCE
*PIBURN, MARVIN F, WICHITA
PICKENS, ANDREW T, WICHITA
PIERCE, CHARLES F, TOPEKA
PIERCE, DONALD R, TOPEKA
PIERCE, GEORGE E, KANSAS CITY
PIERRON, GEORGE J, OLATHE
PIERSON, WEIR, MCPHERSON
PILCHARD, WILLIAM A, SHAWNEE MISSION
PINGLETON, SUSAN K, KANSAS CITY
PINGLETON, WILLIAM WARREN, SHAWNEE MISSION
PINSKER, JACOB A, WICHITA
PIPPIN, LYNNE K, KANSAS CITY
PISCHKE, FRANK J, KANSAS CITY
PITMAN, WILL O, PRATT
PITTS, RONALD L, SHAWNEE MISSION
PIZARRO, FE E, DODGE CITY
PLACEK, DEBRA C, LAWRENCE
PLOWMAN, CARL W, JEWELL
POGSON, GEORGE W, PITTSBURG
POL, P ALBERT, KANSAS CITY
POLING, TERRY L, WICHITA
POLLACK, SIMON, WICHITA
POLLAND, STEPHEN M, D.O., WICHITA
POLLOCK, ANTHONY G A, WICHITA
POLLY, RICHARD E, TOPEKA
POLSON, ROBERT C, GREAT BEND
POMMERENKE, FORREST A, DE SOTO
POOLE, BERNARD T, WICHITA
POONAWALA, HUSENI E, SHAWNEE MISSION
PORTER, DAVID M, KANSAS CITY
PORTER, GARRY L, WICHITA
PORTER, ROBERT D, TOPEKA
POTTER, ROBERT L, KANSAS CITY
POWELL, CAROL W, SHAWNEE MISSION
POWELL, KENNETH A, SHAWNEE MISSION
POWELL, WILLIAM R, TOPEKA
POWELL II, BENSON M, TOPEKA
POWERS, G ROBERT, KANSAS CITY
POWERS, HAROLD W, SUN CITY, AZ
POWERS, K DEAN, WICHITA
PRAEGER, MARK A, LAWRENCE
PRAKALAPAKORN, OARANEE, NESS CITY
PRAKALAPAKORN, YANYONG, NESS CITY
PRAY, CLAUDIA M, LEAVENWORTH
PREHEIM, OELBERT V, NEWTON
PREMSINGH, NALINI G, KANSAS CITY
PRENTICE, WALTER B, INDEPENDENCE
PRENTISS, HAROLD, NEWTON
PRESKORN, SHELDON H, KANSAS CITY
PRESTON, DAVID F, KANSAS CITY
PRESTON, RALPH R, TOPEKA
PRESTON, RICHARD, GREAT BEND
PRETZ, JAMES B, KANSAS CITY
PRICE, HILTON I, KANSAS CITY
PRICE, JAMES GORDON, KANSAS CITY
PRICE, VAUGHAN C, MCPHERSON
PRIETO, JORGE N, KANSAS CITY
PRIETO, LUIS E, WICHITA
PROCHASKA, MARK L, KANSAS CITY
PROCHAZKA, OTTO F, LIBERAL
PROCTOR, ROBERT W, EL DORADO
PROKOP, BRADFORD S, TOPEKA
PRONKO, MICHAEL J, SHAWNEE MISSION
PROUD, G ONEIL, KANSAS CITY
PUGH, DAVID M, KANSAS CITY
PULLMAN, NORMAN K, WICHITA
*PULLUM, RICHARD W, D.O., WICHITA
PURINTON, LEW W, WICHITA
PUTNAM, LYLE B, WICHITA
PYLE, LUCIEN R, TOPEKA

Q

QAMAR, YUSUF, NEWTON
QUACKENBUSH, ROBERT P, SPEARVILLE
QUENZER, RONALD W, PRATT
QUIJANO JR, RAMON S, STAFFORD
QUINN, CHARLES E, KANSAS CITY
QUINONES, ELADIO A, TAMPA, FL

R

RABE, MELVIN A, LEAVENWORTH
RADOVANOV, RADMILA, NEWTON
RAGHAVAN, PARULA P, WICHITA
RAGHAVAN, PRAKASH V, WICHITA
RAGLAND, CHARLES W, KANSAS CITY
RAHMAN, HAFIZ M A, TAMPA, FL
RAINBOW-EARHART, KATHRYN A, TOPEKA
RAJEWSKI, RICHARD L, HAYS
RAJU, A S PADMA, TOPEKA
RALSTIN, JAMES H, SHAWNEE MISSION
*RAMADAN, HOSSEIN, WICHITA
RAMCHANDANI, A P, ULYSSES
RAMIREZ, AUGUSTO H, PITTSBURG
RAMIREZ, IRENE, PITTSBURG
RAMSEY, BARTLETT W, TOPEKA
RANDALL, GEORGE R, WICHITA
RANGLES, MICHAEL J, WICHITA
RANSOELL, EDGAR C, TOPEKA
RANSOM, JAMES H, TOPEKA
RANSOM, WILLARD B, OTTAWA
RARDIN, MARY L, ATCHISON
RATE, PEGGY S, HUTCHINSON
RATE, ROBERT R, HUTCHINSON
RATHBUN, EDWIN D, LIBERAL
RAUSA JR, FRANCISCO C, WICHITA
RAWCLIFFE JR, ROBERT A, WICHITA
RAZEK, ZACK A, WICHITA
READ, WILLIAM T, COFFEYVILLE
READER, G WHITNEY, WICHITA
REALS, WILLIAM J, WICHITA
REAZIN, WALTER L, WICHITA
RECKLING, FREDERICK W, KANSAS CITY
REDDI, RAGHUNATH P, WICHITA
REDDY, B N, HILL CITY
REDDY, EASHWER K, KANSAS CITY
REDDY, P JAGANNADHA, HILL CITY
REDDY, SATTI S, DODGE CITY
REDDY, VENUMBAKA C, EL DORADO
REDFORD, JOHN W B, KANSAS CITY
REDING, DOUGLAS J, KANSAS CITY, MO
REEB, RONALD JOSEPH, KANSAS CITY
REECE, A THOMEN, GARDNER
REECE, RICHARD J, SALINA
REED, A J, WICHITA
REED, O CRAMER, WICHITA
REED, DAVID D, WICHITA
REED, JAMES S, LAWRENCE
REED, JAMES STEWART, KANSAS CITY
REED, RALPH R, LAWRENCE
REESE, JACK D, LIBERAL
REESE, JOHN L, LAWRENCE
REEVES, BRADFORD F, WICHITA
REEVES, CHARLES S, FORT SCOTT
REGIER, LADONNA M, COLBY
REILLY, DOUGLAS K, KANSAS CITY
REINHAROT-WULF, TAISSIA L, GARDEN PLAIN
REINKING, VICTOR E, TOPEKA
REISMAN, MICHAEL ALAN, WICHITA
REITH, PAUL, LAWRENCE
REITZ, LELAND C, MANHATTAN
REITZ, ROGER P, MANHATTAN
REIVICH, RONALD S, SHAWNEE MISSION
RELIHAN, DONALD A, WICHITA
REMPEL, JOHN H, WICHITA
RENDON, HUMBERTO M, AL AIN,
REPLOGLE, CHARLES B, GREAT BEND
RESCHLY, RON, HALSTEAD
REYES JR, FRANCISCO A, OTTAWA
REYMOND, RALPH D, TOPEKA
REYNOLDS, JEFFREY C, HAYS
REYNOLOS, LLOYD W, HAYS
RHOADS, JAMES P, WICHITA
RHODEN, CURTIS H, WICHITA
RHODES, IVAN E, WICHITA
RHODES, JAMES B, KANSAS CITY
RHODES, LOWELL M, WICHITA
RHODES, MARTIN L, KANSAS CITY
RICCI, ROBERT LAWLER, TOPEKA
RICE, BERNARD F, SHAWNEE MISSION
RICE JR, FREDERICK A, KANSAS CITY
RICH, ELDON S, NEWTON
RICH, JOSEPH E, TOPEKA
RICHARDS, DALLAS LEE, HAYS
RICHARDS, DENNIS D, CLAY CENTER
RICHARDS, JON F, PHILLIPSBURG
RICHARDSON, J M, TOPEKA
RICHARDSON, JAY L, KANSAS CITY
RICHARDSON, STEWART F, WICHITA
RICHTER, DON G, SHAWNEE MISSION
RICK JR, GREGORY G, SHAWNEE MISSION
RIDER, JAMES W, ATCHISON
RIEDEL, ROBERT H, TOPEKA

* Probationary members.

RIEDERER, ROBEPT E, WICHITA
RIEGER, ERNEST H, WICHITA
RIEPE, ROGEP E, WICHITA
RIFFEL, LAWRENCE D, SHAWNEE MISSION
RIGLER, WILSON F, ARMA
PILEY, RAY B, KANSAS CITY
RINOT, PHILLIP L, FREDONIA
PINKENBERGER, ROBERT, KANSAS CITY
RIORDAN, HUGH D, WICHITA
RISING, JESSE D, KANSAS CITY
RITCHIE, KAREN S, SHAWNEE MISSION
RIZZA, ROBERT G, HALSTEAD
ROACH, NEIL E, WICHITA
*ROAN, YEAI, WICHITA
ROBERTS, OANIEL K, WICHITA
ROBERTS, RICHARD S, LAWRENCE
ROBEPTS, WARREN E, TOPEKA
*ROBERTS, ROGER W, D.O., WICHITA
ROBEPTSON, EDWARD J, SHAWNEE MISSION
ROBERTSON, JOSEPH K, WICHITA
ROBINSON, OAVID B, TOPEKA
ROBINSON, DAVID W, KANSAS CITY
ROBINSON, EDGAR L, INDEPENDENCE
ROBINSON, G DONALO, WICHITA
ROBINSON, JOHN D, SHAWNEE MISSION
ROBINSON, JOHN E, WICHITA
ROBINSON, MICHAEL O, HAYS
ROBINSON, RALPH G, KANSAS CITY
ROBINSON, ROBERT H, WICHITA
ROBISON, JAMES T, SHAWNEE MISSION
ROBL, DAVID A, WICHITA
ROCHANAYON, PIRA, ELLIS
RODEPICK, JAMES E, SALINA
RODRIGUEZ, ALBERTO, KIOWA
RODRIGUEZ, PAUL L, GAPDEN CITY
RODRIGUEZ, PAUL G, KANSAS CITY
*RODRIGUEZ-RAMOS, EPNEST P, WICHITA
RODRIGUEZTOCKER, LILIA, WICHITA
ROEDER, ROBERT E, TOPEKA
ROEMBACH, JEANINE L, TOPEKA
ROMALIS, BRIAN E, WICHITA
ROMEISER, REX S, SALINA
ROMITO, CYNTHIA L, SHAWNEE MISSION
RCMONDO, STEVEN A, OLATHE
ROOK, LEE E, KANSAS CITY
ROOS, MAUREEN, WICHITA
RORABAUGH, DONALD C, ABILENE
ROSE, DONALD L, BELLA VISTA, AR
ROSE, GRAHAM C, MANHATTAN
ROSE, SHELBY D, WICHITA
ROSEN, OAVID, WICHITA
ROSENBERG, STANTON L, SHAWNEE MISSION
ROSENBERG, THOMAS F, WICHITA
ROSENTHAL, PICHARD, SHAWNEE MISSION
ROSENTHAL, STANTON J, KANSAS CITY
ROSS, DAVID K, ARKANSAS CITY
ROSS, DENNIS LEE, WICHITA
ROSS, JACK L, TOPEKA
ROSS, STEVEN E, KANSAS CITY
ROSSITTO, ANTHONY F, SAN FRANCISCO, CA
ROTERT, LAPPY, TOPEKA
ROTH, ALAN E, KANSAS CITY
ROTHSTEIN, TERRY B, PARSONS
POWLETT, JACK G, PAOLA
ROY, WILLIAM P, TOPEKA
RUBIN, HEPBERT M, SHAWNEE MISSION
PUBIN JR, BEN, KANSAS CITY
PUBLE JR, JAMES L, OVERBROOK
PUCKER, CLEMENS, TOPEKA
RUEB, ANDREW E, SALINA
RUHLEN, JAMES L, OLATHE
RUIZ, CAPLOS M, GREAT BEND
RUMOLD, MERVIN J, SHAWNEE MISSION
PUNNELS, JOHN B, TOPEKA
RUPP, PICHAPD J, TOPEKA
PUSSELL, PHILIP W, WICHITA
RUSSO, LIBBIE V, KANSAS CITY
PUTH, WILLIAM E, KANSAS CITY
RUTNGAMLUG, LUECHA, HAYS
RUZICKA, LAWRENCE J, CONCOPOIA
RYAN, MICHAEL E, SHAWNEE MISSION
RYAN, MICHAEL J, KANSAS CITY
RYAN, W SCOTT, EMPORIA
RYMER, POBERT A, SHAWNEE MISSION

S

SABIN JR, GEORGE M, WICHITA
SACHEN, FREDERICK L, SHAWNEE MISSION
SADIQ, SULEMAN, WICHITA
SAEED, MOHAMMAD, WICHITA
SAFFO, KAPL S, SHAWNEE MISSION
SALEH, GEORGE A, D.O., KANSAS CITY, MO
SAMUEL, CHANDY C, WINFIELD
SAMUEL, SHANTHI, WINFIELD
SANCHEZ, ROGELIO, TOPEKA
SANDBERG, CHRIS B, EL DORADO
SANDERS, GLORIA DEANNA, WICHITA
SANDERS, J ALAN, LAWRENCE
SANDHU, JAGVEEP SINGH, WICHITA
SANDHU, PAUL S, COFFEYVILLE
SANTOSCOY, GILBERT S, WICHITA
SARGENT, JOSEPH O, TOPEKA
SATHYANARAYANA, SAPASWATHI, SHAWNEE MISSION
SAUL, F WILLIAM, EMPORIA
SAUNDERS, MICHAEL E, CONCORDIA
SAVIN, VIRGINIA J, KANSAS CITY
SAWKAR, LAXMIDAS A, SHAWNEE MISSION
*SAYEED, BASEER A, HAYSVILLE
SAYLER, JEPCME, GREAT BEND
SAYLOR, EDWARD H, TOPEKA
SAYLOR, LESLIE L, TOPEKA
SAYLOR, MARK, TOPEKA
SAYLOR, STEPHEN, TOPEKA
SCALES, WILLIAM M, HUTCHINSON
SCAMMAN, W WIKE, TOPEKA
SCANLAN, TIMOTHY M, SALINA
SCHAEFER, JOSEPH PETEP, SHAWNEE MISSION
SCHAFFER, CLARENCE K, SANTA CRUZ, CA
SCHEEL, BRADLEY J, HUTCHINSON
SCHELLINGER, RICHARD P, EMPORIA
SCHEPER, ALFRED L, TRUTH OR CONSEQ, NM
SCHILTZ, FRANCES, WICHITA
SCHIMKE, R NEIL, KANSAS CITY
SCHLACHTER, ERNEST P, WICHITA
SCHLEMMER, ROGER B, PITTSBURG
SCHLICHER, JOHN E, WICHITA
SCHLOESSER, HARVEY L, TOPEKA
SCHLOESSER, PATRICIA T, TOPEKA
SCHLOTTERBACK, WILLIAM E, BELLEVILLE
SCHLUETER, JOHN J, WICHITA
SCHMAUS, LYLE F, IOLA
SCHMEIOLEP, OAVID ALLEN, ARKANSAS CITY
SCHMIOT, HERBERT R, NEWTON
SCHMIOT, RAMON WARNER, SALINA
SCHNELLE, JOACHIM, WICHITA
SCHNOEBELEN, RENE E, KINSLEY
SCHNOSE, GREGORY D, LAWRENCE
SCHOPF, CLIFTON C, WICHITA
SCHOTLAND, EDWARD S, KANSAS CITY
SCHRAM, PETER CHAPLES, TOPEKA
SCHREPFER, POSEMARY, KANSAS CITY
SCHROEDEP, SYONEY O, LAWPENCE
SCHROLL, JACK C, HUTCHINSON
SCHROLL, JOHN T, SHAWNEE MISSION
SCHUETZ, PERRY N, GPEAT BEND
SCHUKMAN, JAY S, GPEAT BEND
SCHULTZ, JAMES E, COUNCIL GROVE
SCHURLE, DALE R, MCPHERSON
SCHWARTING, J STEVE, ABILENE
SCHWARTZ, EUGENE W, DODGE CITY
SCHWARTZ, V OEAN, WICHITA
SCHWEGLER, PAYMOND A, LAWRENCE
SCHWEGLER, RAYMOND A, KANSAS CITY
SCHWOPM, CURTIS P, KANSAS CITY
SCLAP, WILLIAM C, SHAWNEE MISSION
SCOTT, ALEX, JUNCTION CITY
SCOTT, CHESTER E, SALINA
SCOTT, DUANE L, BELLEVILLE
SCOTT, VINCENT L, WICHITA
SCOTT, WILLIAM H, WICHITA
SEAMAN, LAUREN I, OLATHE
SEBREE, STEVEN G, SALINA
SEELEY, JAMES C, HOLTON
SEGERSON, JOHN A, TOPEKA
SEGLIE, FLOYD RONALO, PITTSBURG
SEHDEV, JOAN, TOPEKA
SEITZ JR, JOSEPH E, ELLSWOPTH
SEKAVEC, GORDON B, OAKLEY
SEN SARMA, PRONAB K, WICHITA
SEREPES, EDGAR P, KANSAS CITY
SETTLE JR, RUSSELL O, OLATHE
SETTLE SR, RUSSELL O, TOPEKA
SEVIFP, SAMUEL M, MUSKOGEE, OK
SHAAD, DOROTHY J, SHAWNEE MISSION
SHAFER, PRESTON J, WICHITA
SHAFFER, KATHLEEN BRAY, SHAWNEE MISSION
SHAFFER, STANLEY G, SHAWNEE MISSION
SHAH, ASHOK H, WINFIELD
SHAH, MIAN, LARNED
SHAH, MUKHTAR H, WICHITA
SHAH, NASPEEN, LARNED
SHAH, SHAPFUDDIN, HALSTEAD
SHANKER, STUART G, OLATHE
SHAPIPO, MAPTIN, ELLINWOOD
SHARMA, APUN L, PARSONS
SHAW, JOSEPH L, TOPEKA
SHAW, RICHARD C, WICHITA
SHAW JR, JAMES W, HUTCHINSON
SHEAFOR, DOUGLAS, TOPEKA
SHEARS, ROBERT N, HUTCHINSON
SHEERN, MAPK DOUGLAS, ABILENE
SHEFFER, KEITH D, OLATHE
SHEHI, LORA J, WICHITA
SHELLITO, JOHN G, WICHITA
SHELTON, STEPHEN E, TOPEKA
SHEPARD, LEROY W, LARNED
SHEPPARD, ROBERT G, SMITH CENTER
SHERIDAN, RANDY M, SHAWNEE MISSION
SHERMAN, POBERT P, KANSAS CITY
SHERWOOD JR, CLAPENCE E, TOPEKA
SHEU, W ERIC, TOPEKA
SHIELD, CHAPLES, WICHITA
SHIELOS, THOMAS M, MANHATTAN
SHIELDS JR, JAMES M, EL DOPADO
SHIFLET, ALBERT W, WHEELING, MO
SHIVEL, DAVID G, GREAT BEND
*SHOFFNER, RICHARD W, WICHITA
SHOFSTALL, WILLIAM H, SHAWNEE MISSION
SHORTES, LOIS E, FT BRAGG, CA
*SHPADEP, C ERIC, WICHITA
SHRADER, DOYLE A, WICHITA
*SHURTZ, GLEN L, WICHITA
SHUSS, JOHN L, GAPDEN CITY
SIBALA, JUSTO L, PRATT
SIEGEL, ALBERT R, WICHITA
SIEMENS, PICHARD A, LYONS
SIFERS, EARL C, KANSAS CITY
SIFERS, TIMOTHY M, KANSAS CITY
SIFFORD, R LAWRENCE, WICHITA
SILER, EUGENE T, HAYS
SILLS, CHARLES T, NEWTON
SILLS, THOMAS O, SHAWNEE MISSION
SILVEP, BRADD J, SHAWNEE MISSION
SILVERS, ALVIN, KANSAS CITY
SIMMONS, ROBERT EAPLE, NEWTON
SIMPSON, ROBERT LIMBAUGH, OBERLIN
SIMPSON, TOM C, STERLING
SIMPSON, WILLIAM S, TOPEKA
SINGER, PHILIP A, KANSAS CITY, MO
SINGH, GIRVAR, ARKANSAS CITY
SINNING, GAPY, HIAWATHA
SISK, PHILLIP B, TOPEKA
SIWEK, CHRISTOPHER W, EL ODRADO
SKAER, STANLEY ALLEN, EUREKA
SKIBBA, RICHARD M, WICHITA
SKIKNE, BARPY S, KANSAS CITY
SLEEPER, CAPOL A, KANSAS CITY, MO
SLCO, MILO G, SALINA
SLUTSKY, LAWRENCE JOEL, WICHITA
SMITH, ALVIN L, WICHITA
SMITH, BOYD E, SALINA
SMITH, BRUCE G, ARKANSAS CITY
SMITH, DALE C, SHAWNEE MISSION
SMITH, DAVID E, SALINA
SMITH, DONALD J, SHAWNEE MISSION
SMITH, HAPOLD R, SALINA
SMITH, JOHN D, LAPNED
SMITH, LED A, TOPEKA
SMITH, NEWTON C, ARKANSAS CITY
SMITH, PERRY MILTON, GREAT BEND
SMITH, THOMAS WILLIAM, HUTCHINSON
SMITH, TIMOTHY WM, WICHITA
SMITH, WILLIAM P, KANSAS CITY
SMITH JR, FLOYD L, COLBY
SMITH JR, WILLARD J, WICHITA
SMITH-HOLMES, BARBARA K, KANSAS CITY, MO
SNARR, JACK W, TOPEKA
SNODELL, FIRMIN E, SHAWNEE MISSION
SNODGRASS, WAYNE R, KANSAS CITY
SNOOK, ROBERT RUFUS, MCLOUTH
SNOW, DONALD L, LANSING
SNOW JR, ARTHUR D, SHAWNEE MISSION
SNOWBARGEP, MARVIN O, EMPORIA
SNYDER, C JOHN, WINFIELD
SNYDEP, GREGG M, WICHITA
SNYDER, HOWARD E, WINFIELD
SNYDER, THOMAS E, SALINA
SNYDER JP, RICHARD HENRY, OLATHE
SOELDNER, JAMES OLIVER, SHAWNEE MISSION
SOHN, YUNGGYO, TOPEKA
SOLOMON, HERMAN, WICHITA
SOLTZ, POBERT A, WICHITA
SOMEPS, MARVIN M, WICHITA
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A	Allergy
ADM	Administrative Medicine
ADT	Addictionology
AM	Aviation Medicine
ANES	Anesthesiology
CD	Cardiovascular Disease
CDS	Cardiovascular Surgery
CDTS	Cardiovascular & Thoracic Surgery
CHN	Child Neurology
CHP	Child Psychiatry
CP	Clinical Pharmacology
D	Dermatology
DR	Diagnostic Roentgenology
EENT	Eye, Ear, Nose and Throat
EM	Emergency Medicine
END	Endocrinology
ENT	Ear, Nose, Throat
ES	Endoscopy Surgery
FM	Family Medicine
FP	Family Practice
GE	Gastroenterology
GEN	Genetics
GER	Geriatrics
GP	General Practice
GPM	General Preventive Medicine
GPVS	General & Peripheral Vascular Surgery
GS	General Surgery
GYN	Gynecology
HEM	Hematology
ID	Infectious Diseases
IE	Insurance Examination
IM	Internal Medicine
MDST	Medical Student
MFM	Maternal Fetal Medicine
MO	Medical Oncology
N	Neurology
NEP	Nephrology
NM	Nuclear Medicine
NEO	Neonatology
NP	Neuropsychiatry
NR	Nuclear Radiology
NS	Neurological Surgery
OBG	Obstetrics and Gynecology
OM	Occupational Medicine
ON	Oncology
OPH	Ophthalmology
ORS	Orthopedic Surgery
OS	Other Specialties
OST	Osteopathy
OTO	Otorhinolaryngology
P	Psychiatry
PATH	Pathology
PD	Pediatrics
PDA	Pediatric Allergy
PDC	Pediatric Cardiology
PDE	Pediatric Endocrinology
PDN	Pediatric Neurology
PDO	Pediatric Ophthalmology
PDR	Pediatric Radiology
PDS	Pediatric Surgery
PGER	Psychogerontology
PH	Public Health
PM	Physical Medicine & Rehabilitation
PNP	Pediatric Nephrology
PS	Plastic Surgery
PUD	Pulmonary Disease
PYM	Psychosomatic Medicine
R	Radiology
RES	Resident
RHU	Rheumatology
RM	Rehabilitative Medicine
RN	Radiology & Neurology
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RT	Radiation Therapy
SM	Sports Medicine
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U	Urology
00	Retired

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JACKSON, VICTOR L, BOX 467,67330
784-5318
20 M 2105 50 FP

ANDALE—316
(Sedgwick County Society)

GOODWIN, MARY K, ANDALE CLINIC,67001
267-0865
53 F 1902 77 FP
STECH, JOSEPH M, ANDALE CLINIC,67001
445-2152
27 M 3006 56 FP

ANDOVER—316
(Sedgwick County Society)

KLINGMAN, DIANE O, PO BOX 426,67002
733-1331
53 F 1902 79 FP

ANTHONY—316
(Tri-County Society)

ANTRIM, PHILIP JENIFER, 1101 E SPRING,67003
842-5144
15 M 1902 42 FP
HALL, JERRY O, 1101 E SPRING,67003
842-5144
31 M 3901 56 FP

ARKANSAS CITY—316
(Cowley County Society)

ALVAREZ, NORBERTO, 112 E CENTRAL,67005
442-4850
27 M 27501 53 FP
AUCAR, ALFREDO, BOX 1105,67005
442-1710
23 M 27501 53 OTO
BAKER, ROBERT R, O.D., 2508 EDGEMONT BOX 714,67005
442-8924
50 M 2878 78 FP
CAMPBELL, GARLAND L, 114 W WALNUT,67005
442-1350
13 M 1902 40 U
GREEN, LAWRENCE C, PO BOX 173,67005
442-4850
43 M 3901 69 FP
HINSHAW, EDGAR O, RT #3,67005
442-1273
15 M 1902 51 R
LEIKER, JOSEPH, 112 E CENTRAL,67005
442-7900
48 M 1902 73 IM
MARVEL, JAMES EBBERT, 2508 EDGEMONT,67005
442-0222
43 M 3901 72 ORS
OLD, JERRY L, PO BOX 1148,67005
442-2100
49 M 1902 74 FP
PEREIRA, WILLY G, 2508 EDGEMONT DR,67005
442-8540
39 M 73701 67 IM
ROSS, DAVID K, PO BOX 1148,67005
442-2100
48 M 1902 75 FP
SCHMEIDLER, DAVID ALLEN, 510 W RADID LANE,67005
442-2100
54 M 1902 79 FP
SINGH, GIRVAR, 2508 EDGEMONT DR,67005
442-4300
40 M 49555 64 OPH
SMITH, BRUCE G, 115 E RADIO LANE,67005
442-5600
20 M 1902 44 IM
SMITH, NEWTON C, PO BOX 1148,67005
442-2100
21 M 3901 45 FP
YOACHIM, ROBERT W, 510 W RADIO LANE,67005
442-2100
52 M 3005 78 FP

ARMA—316
(Crawford County Society)

RIGLER, WILSON F, 511 WASHINGTON,66712
347-8619
42 M 1803 69 FP

ASHLAND — 316
(Iroquois County Society)

BOWEN, JAMES D., PO BOX 876-620 HIGHLAND, 67831
635-2795
39 M 52 66 FP

ATCHISON—913
(Atchison County Society)

BOSSE, FRANK K, 1301 RIVERVIEW DRIVE,66002
-
09 M 2802 33 00
BRADY, CHARLES S, PO BOX 245,66002
367-1232
11 M 3006 38 GS
BRIBACH, EUGENE J, 125 1/2 N 5TH,66002
367-0225
83 M 2802 05 OPH
BROWN, ROBERT O, 1400 NORTH 2ND,66002
367-1922
14 M 1902 44 FP
BURKE, JOSEPH V, 1301 N 3RD,66002
367-5496
35 M 3006 66 GS
DUNLAP, MICHAEL O, 1225 N SECOND,66002
367-0362
53 M 1902 79 FP
EPLEE, JOHN R, 1225 N SECOND,66002
367-0362
53 M 1902 78 FP
FAST, ROBERT E, 1225 N 2ND,66002
367-0362
48 M 1902 74 OBG
FAST, W SPENCER, RAMSAY MED CLINIC,66002
367-0362
11 M 3006 39 FP
GROWNEY, JOHN T, 801 ATCHISON,66002
367-5020
37 M 3006 63 FP
HART, LAWRENCE E, 1412 N 2ND,66002
367-5054
32 M 1902 64 FP
MESROPIAN, GEORGE D, 1225 N SECOND,66002
367-1114
48 M 60501 74 GS
MORRISON, IRA R, 825 N 10TH,66002
-
07 M 1611 36 1M
RAROIN, MARY L, MOUNT ST SCHOLASTICA,66002
-
52 F 1902 77 FP
RIDER, JAMES W, 1225 N 2ND ST,66002
367-0362
47 M 2803 73 FP
TIVORSAK, ARKOM, 1716 COUNTRY LANE,66002
367-2131
40 M 89101 68 R
WALLACE JR, WAYNE O, 1301 N THIRD,66002
367-7300
36 M 2803 65 FP
WULFF, EDWIN T, 923 N FIFTH,66002
367-5033
07 M 2834 36 FP
YOUNG, CHARLES H, 1301 N 3RD,66002
367-4053
23 M 1902 53 FP

ATTICA—316
(Tri-County Society)

CRANDALL, DORA, 710 N LOGAN,67009
254-7237
39 F 64914 81 GP
STONE, GRANT C, 500 N HARPER,67009
254-7219
08 M 5605 35 FP

ATWOOD—316
(Northwest Kansas Society)

DILL, ROONEY, PO BOX 67,67730
626-3229
41 M 74811 77 GP

AUGUSTA—316
(Butler-Greenwood Society)

ANDERSON, DALE W, 120 W JOSEPHINE,67010
775-5432
30 M 1902 55 FP
BARBER, JAMES L, 120 W JOSEPHINE,67010
775-5432
31 M 1902 57 FP
TUONG, TRAN MANH, 120 W JOSEPHINE,67010
775-5432
39 M 94101 65 FP

BAXTER SPRINGS—316
(Cherokee County Society)

ALQUIST, VERYL D, 21ST & FAIRVIEW,66713
623-4942
17 M 1902 42 GS
CHUBB, RICHARD M, 445 EAST 10TH,66713
856-2144
29 M 1606 54 FP

BELLEVILLE—913
(Republic County Society)

BEIDERWELL, PAUL L, 2703 M ST,66935
-
08 M 3901 38 FP
DOUBEK, HERBERT D, PO BOX 250,66935
527-2237
28 M 1902 56 FP
SCHLOTTERBACK, WILLIAM E, 1202 L,66935
378-3511
31 M 1902 61 FP
SCOTT, DUANE L, BELLEVILLE CLINIC,66935
527-2217
34 M 1902 60 FP
WARD, JAMES A, 1206 18TH ST,66935
527-2217
34 M 1902 58 FP

BELOIT—913
(Mitchell County Society)

DOBRATZ, ROBERT A, 310 W 8TH,67420
738-2246
24 M 1902 52 FP
DRAKE, DOUGLAS J, MEDICAL CENTER,67420
738-2246
43 M 1902 71 FP
KLENDA JR, MARTIN B, BELOIT MED CENTER,67420
738-2246
38 M 1643 63 GS
LONEY, JOHN M, 310 WEST 8TH,67420
738-2246
50 M 1902 74 1M
WELTMER, ROGER P, 112 W MAIN,67420
738-2574
18 M 1902 44 U

BLUE RAPIDS—913
(Northeast Kansas Society)

LAWLESS, HAROLD L, 607 LINCOLN,66411
226-7202
29 M 702 54 FP

BONNER SPRINGS—913
(Wyandotte County Society)

FUNK, EDWARD O, 311 W HILL RD,66012
-
04 M 1902 41 ANES
MAY, KENNETH L, 122 N NETTLETON,66012
422-2020
20 M 1902 51 FP
MITTS, ERNEST W, 122 N NETTLETON,66012
422-2020
22 M 1902 51 FP
WAGGONER, FRANKLIN E, 122 N NETTLETON,66012
422-2020
26 M 1902 61 FP

BUHLER—316
(Reno County Society)

FRIESEN, ORLANOO J, 107 W 2ND,67522
543-2330
27 M 1902 56 FP

BURLINGAME—913
(Shawnee County Society)

GOERING, EMIL L, 130 W SANTA FE,66413
654-2300
27 M 1902 57 1M

CALDWELL—316
(Tri-County Society)

KINNAN, L F, 523 S MARKET,67022
845-6422
18 M 3901 42 FP

CANEY—316
(Southeast Kansas Society)

MOORE, ROBERT F, 4TH & MCGEE,67333
879-2135
28 M 1902 56 FP

CEDAR VALE—316
(Southeast Kansas Society)

COHNBERG, ROSELLEN E, PO BOX 398,67024
758-2266
22 F 2802 47 FP

CHANUTE—316
(Southeast Kansas Society)

ABBUEHL, OON R, 505 SCUTH PLUMMER,66720
431-2500
18 M 1902 44 GS
ASHLEY, SAMUEL G, 505 SOUTH PLUMMER,66720
431-2500
16 M 1902 43 FP
BAKER, HENRY K, 1220 W FOURTH,66720
431-1600
08 M 1606 35 GS
BURKMAN, REUBEN J, 1501 W 7TH,66720
431-9310
28 M 1902 54 FP
DICK JR, HENRY J, 1501 W 7TH,66720
431-9310
27 M 1902 58 FP
GAREY, WILLIAM JOHN, PO BOX 321 RT 2,66720
431-4000
44 M 6501 70 OR
GEHRT, EARL B, 505 SO PLUMMER,66720
431-2500
32 M 1902 62 FP
HASKINS, ROBERT J, 505 S PLUMMER,66720
431-2500
46 M 1902 74 FP
KIHM, ALBERT A, 505 S PLUMMER,66720
431-2500
27 M 1902 55 FP
MIH, ALEXANOER, 1002 WEST 4TH,66720
473-2227
22 M 24209 47 ANES
PARHAM, VERODN W, 505 S PLUMMER,66720
431-2500
47 M 1902 73 FP
VAN HOUOEN, CHARLES E, 505 S PLUMMER,66720
431-2500
52 M 1902 77 GS

CHAPMAN—913
(Dickinson County Society)

SVOBODA, CHARLES R, 413 N MARSHALL,67431
922-6400
18 M 1902 46 FP

CHETOPA—316
(Labette County Society)

PEFFLY, ELMER D, 327 MAPLE,67336
236-7188
22 M 3901 53 FP

CLAY CENTER—913
(Clay County Society)

ANOERSON, SEVERT A, 1749 BERGLUNO OR,67432
-
07 M 1902 36 00
BOGNER, PAUL F, 1318 SUNRISE CIRCLE,67432
632-2185
52 M 1902 77 GS
CARLETON, RICHARO C, 709 LIBERTY,67432
632-5603
31 M 3005 61 FP
OALUM, PETER JOSEPH, 709 LIBERTY SUITE C,67432
632-2181
45 M 2803 76 FP
GOOO, MICHAEL W, 709 LIBERTY,67432
632-2181
53 M 1902 78 FP
MCVAY, R BRUCE, 1503 FIFTH ST,67432
-
97 M 1902 29 00
O'OONNELL, RICHARO H, 709 LIBERTY,67432
632-3101
16 M 1902 41 GS
RICHAROS, DENNIS D, 115 S 6TH,67432
632-5621
34 M 1902 60 FP
STEINBAUER, JEFFREY ROBERT, 709 LIBERTY,67432
632-2181
51 M 1902 77 FP

CLYDE—913
(Cloud County Society)

FREEBORN JR, WARREN S, ,66938
446-2221
26 M 1720 51 FP

COFFEYVILLE—316
(Southeast Kansas Society)

BANKS, GILBERT, COFFEYVILLE DOCTORS' CL,67337
251-7500
49 M 2401 75 1M
CAMPBELL, WILLIAM H, 1411 W 4TH,67337
251-3235
39 M 1902 65 DPH
COYLE, JOHN F, PO BOX 487,67337
251-2400
21 M 1902 44 FP
OICKINSON, CHARLES R, 108 W 7TH,67337
251-1340
20 M 1606 44 GS
OIXON, RAYMOND W, 1411 W 4TH,67337
251-1090
46 M 4706 71 GS
EOROZO, M LUZ LUNA, PO BOX 856,67337
251-1200
F 74801 68 PATH
ELLIS, STEPHEN S, 111 N HIGHLANO,67337
-
11 M 2802 36 00
GIBBS, EUGENE, PO BOX 716,67337
251-7260
M 64914 68 FP
HA, SANG W, 504 WILSHIRE,67337
251-7750
35 M 58309 60 ORG
HAN, CHAN S, 908 SIGGINS,67337
251-1560
35 M 58306 61 PO
HOWERTER JR, BERNARD E, PO BOX 659,67337
251-4790
43 M 1803 68 U
JIRICKO, MILOS, 2510 W EIGHTH,67337
251-1200
37 M 28601 61 ANES
NICHOLS, RICHARO, 1411 W FOURTH,67337
251-6540
M 1803 73 ORS

READ, WILLIAM T, 1411 WEST 4TH,67337
251-1120
16 M 2802 40 FP
SANDHU, PAUL S, PD BOX 257,67337
251-2450
42 M 49508 65 GS
UY, WILSON D, COFFEYVILLE MEM HOSPITAL,67337
251-1200
42 M 74801 67 PATH
VAKAS, JOHN L, 1508 W 4TH,67337
251-3443
38 M 1902 64 IM
WHITE, DONALD C, PD BOX 262,67337
251-1200
35 M 3515 65 R

COLBY—913
(Northwest Kansas Society)

DAHL, ASHER W, COLBY CLINIC,67701
462-3333
28 M 1902 58 FP
HASSETT, GERARD R, 1875 HARVEY,67701
-
24 M 3006 50 R
HILDYARD II, VICTOR H, BOX 28,67701
462-3332
47 M 702 73 FP
JACOBSEN, DWIGHT SKINNER, COLBY CLINIC,67701
462-3333
31 M 3545 60 GS
REGIER, LADONNA M, COLBY CLINIC,67701
462-3332
47 F 1902 73 FP
SMITH JR, FLOYD L, COLBY CLINIC,67701
462-3333
20 M 1902 44 FP

COLDWATER—316
(Iroquois County Society)

GDERING, DONALD D, BOX 74B,67029
582-2136
31 M 1902 56 FP

COLUMBUS—316
(Cherokee County Society)

ANDREASEN, RAYMOND L, 219 S KANSAS,66725
429-3744
47 M 1720 74 FP
BELCHER, GEORGE D, BOX 309,66725
429-2557
34 M 1902 60 FP
JONES, FORREST H, 219 S KANSAS,66725
429-3744
25 M 1902 54 FP
PASIMIO, ROGER S, R2 BOX 259,66725
429-1977
38 M 74801 62 GS

COLWICH—316
(Sedgwick County Society)

LIES, BARTHEL N, 309 S THIRD,67030
-
11 M 2802 37 OD

CONCORDIA—913
(Cloud County Society)

BRAY, AVIS PAGE, 1010 3RD AVE,66901
243-1560
17 F 702 54 FP
BUTT, MUHAMMAD, CONCORDIA MED GROUP,66901
243-1560
46 M 70401 69 GS
COULTER, THAYNE A, D.O., 11TH & WASHINGTON,66901
243-4455
12 M 2878 37 GP
FOWLER, WAYNE L, CONCORDIA MED GROUP,66901
423-1560
23 M 1720 47 IM
GELVIN, E RAYMOND, 835 WEST 9TH,66901
243-1560
03 M 3005 27 GS
GRANT, MICHAEL D, CONCORDIA MED GROUP,66901
243-1560
46 M 1902 79 FP
HOFER, DEWAYNE D, COUNTRY CLUB DR,66901
243-1263
36 M 1606 62 R
KOSAR, CLARENCE D, BOX 362,66901
-
98 M 1902 26 DO
LAWTON, MARVIN K, CONCORDIA MED GROUP,66901
243-1560
31 M 3005 58 GS
LLOYD, JAMES W, 910 WEST 11TH,66901
243-7011
44 M 1902 73 FP
MOORE, JAMES E, PD BOX 305,66901
243-7636
48 M 1902 74 P
NELSON, PAUL L, 1010 THIRD AVE,66901
243-1560
27 M 1902 55 PD
NEWMAN, CARL T, PD BOX 587,66901
243-2511
49 M 64914 76 U
OWENSBY, L C, 222 W SIXTH,66901
243-3386
21 M 2802 44 DPH
RUZICKA, LAWRENCE J, 1115 HILLSIDE DR,66901
243-1560
13 M 3005 40 ANES
SAUNDERS, MICHAEL E, 1404 CRESCENT LANE,66901
243-1560
50 M 1902 73 IM
STRYKER JR, HENRY B, 717 FIRST AVE,66901
-
19 M 3501 44 DD
THORNTON JR, FOXHALL P, 723 W 7TH ST,66901
243-1560
25 M 5101 50 IM
WOLF, PATRICK G, 1413 COUNTRY CLUB DR,66901
243-1560
52 M 1902 77 IM

COTTONWOOD FALLS—316
(Flint Hills Society)

BROWNING, JIMMIE L, BOX 486,66845
273-6753
50 M 1902 78 FP
MCKEE, LEO F, ,66845
273-6681
16 M 1902 39 FP

COUNCIL GROVE—316
(Flint Hills Society)

ALLRED, CHARLES T, 221 HOCKADAY,66846
767-5126
53 M 1902 78 FP
BARKER, ROYAL A, 221 HOCKADAY,66846
767-5126
21 M 1902 53 FP
BLACKBURN, ROBERT W, 221 HOCKADAY,66846
767-5126
22 M 1902 49 FP
SCHULTZ, JAMES E, 221 HOCKADAY,66846
767-5126
26 M 1902 56 FP

CUNNINGHAM—316
(Ninnescah Society)

ALLBRITTEN JR, FRANK F, PD BOX 177,67035
-
14 M 4101 38 DO

DE SOTO—913
(Johnson County Society)

POMMERENKE, FORREST A, 206 E SECOND,66018
585-1177
48 M 1902 75 FP

DENTON—913
(Northeast Kansas Society)

YODER, EMERSON O, ,66017
359-6531
14 M 1902 49 FP

DERBY—316
(Sedgwick County Society)

MCKERRACHER, ROBERT O, 400 A NORTH BALTIMORE,67037
688-1779
27 M 3901 55 FP
MILLER, LAWRENCE H, 606 MULBERRY,67037
788-3741
40 M 1001 67 FP
MOYER, HERMAN J, 200 S BALTIMORE,67037
788-1484
24 M 3901 55 FP
VINZANT, MARK N, 1410 N WOODLAWN,67037
681-3092
45 M 64914 75 FP

DIGHTON—316
(Southwest Kansas Society)

VON LEONROD JR, GEORGE, PO BOX 146,67839
397-5314
16 M 1902 43 FP

DODGE CITY—316
(Ford County Society)

AMAWI, MOHAMMAD S, DODGE CITY MED CENTER,67801
225-1371
46 M 87501 71 GS
AVILA, OSCAR A, DODGE CITY MED CENTER,67801
225-1371
41 M 17603 69 IM
AYUTHIA, ISSARA I, 2004 FREDERICK DR,67801
-
40 M 89101 66 PATH
BAUM, ARNOLD H, DODGE CITY MED CENTER,67801
225-1371
16 M 2101 44 OBG
BETTERTON, DALE C, PO BOX 1413,67801
225-0200
38 M 2604 73 FP
BOLES, R DALE, DODGE CITY MED CENTER,67801
225-1371
23 M 3901 53 PD
BROWNRIGG, RICHARD L, 108 ROSS BLVD,67801
225-0075
35 M 1902 61 U
BUSCH, ANTHONY B, SOUTHWEST CLINIC,67801
-
05 M 2002 31 FP
CHOTIMONGKOL, ANUPONG, DODGE CITY MED CENTER,67801
225-1371
43 M 89102 69 OBG
CONARD, CLAIR C, DODGE CITY MED CENTER,67801
225-1371
27 M 1902 55 IM
GARCIA, GUILLERMO D, 2020 CENTRAL,67801
225-1371
43 M 23101 68 ORS
GREENBERG, GEORGE E, 1904 BURR PARKWAY,67801
276-8241
42 M 401 68 R
JAMBOR, CHRISTOPHER N, DODGE CITY MED CENTER,67801
225-1371
51 M 1902 78 PD
JOHNSON, HOWELL D, DODGE CITY MED CENTER,67801
225-1371
45 M 1902 71 IM
MATTA, JOSE R, 806 SECOND AVE,67801
227-3141
46 M 64914 76 OBG
MCCOY, RONALD, SOUTHWEST CLINIC,67801
227-3141
19 M 3901 47 FP
MCELHINNEY, CHARLES F, DODGE CITY MED CENTER,67801
225-1371
36 M 1902 62 GS
MCGINNIS, MICHAEL J, SOUTHWEST CLINIC,67801
227-3141
41 M 5501 69 GS
MIMOSO, JOSE J, SOUTHWEST CLINIC,67801
227-3141
37 M 4201 61 OBG
NIXON, JAMES E, 2002 FIRST AVE,67801
225-1033
40 M 4812 72 OR
OHMAN, RICHARD J, 1810 1/2 FAIRWAY DR,67801
-
15 M 2407 41 OD
PIZARRO, FE E, 806 SECOND,67801
227-3141
44 F 74810 68 PD
REDDY, SATTI S, 108 RUSS BLVD,67801
225-0075
35 M 49504 66 U
SCHWARTZ, EUGENE W, 1ST NATL BANK BLDG,67801
225-4261
24 M 1902 50 OPH
STOCKWELL, MORGAN D, 2020 CENTRAL,67801
225-1371
24 M 1902 55 IM
TREKELL, WILLIAM V, SOUTHWEST CLINIC,67801
227-3141
25 M 1902 52 ORS
TROTTER, ROGER COURTNEY, 1111 SIXTH AVE,67801
225-6120
47 M 1902 74 FP
VIERTHALER, CARL A, DODGE CITY MED CENTER,67801
225-1371
53 M 1902 78 IM
WILLIAMS, EVAN ROBERT, DODGE CITY MED CENTER,67801
225-1371
25 M 1606 52 GS
ZACHARIAS, CARL KURT, DODGE CITY MED CENTER,67801
225-1371
21 M 40707 47 ORS

EL DORADO—316
(Butler-Greenwood Society)

BRIAN, ROBERT M, 123 N ATCHISON,67042
321-1230
02 M 1606 30 FP
DELLETT, KENNETH B, 3RD & VINE,67042
321-1910
30 M 1902 55 OPH
GIROD, CHARLES I, 123 N ATCHISON ST #103,67042
321-4981
11 M 4706 44 GS
HAFFNER, WILLIAM N, 123 N ATCHISON,67042
321-5049
35 M 1902 61 GS
JACOB, KANNAMPALLY L, 123 N ATCHISON,67042
321-0056
31 M 49537 59 U
KASSEBAUM, GLEN E, 123 N ATCHISON,67042
-
98 M 1606 23 GS
LEE, YONG D, 123 N ATCHISON,67042
321-5630
35 M 58310 60 GS
OLSEN, PHILLIP S, 123 N ATCHISON,67042
321-2100
46 M 1902 73 IM
OVERHOLSER, NORMAN H, 119 N JONES,67042
321-2010
16 M 1902 41 FP
PROCTOR, ROBERT W, 123 N JONES,67042
321-2010
38 M 1902 63 FP
REDDY, VENUMBAKA C, ROOM 302,67042
321-3300
46 M 49511 70 IM
SANDBERG, CHRIS B, 119 NORTH JONES,67042
321-2010
48 M 1902 74 FP
SHIELDS JR, JAMES M, 119 N JONES,67042
321-2010
18 M 4802 42 FP
SIWEK, CHRISTOPHER W, 123 N ATCHISON,67042
321-5211
48 M 75911 71 ORS
WHITE II, BENJAMIN E, 119 N JONES,67042
321-2010
27 M 1902 54 FP

ELKHART—316
(Seward County Society)

CABRERA, ALBERTO, BOX 997,67950
697-2155
30 M 74801 55 GS

1WAY, BELINO D, 411 SUNSET BOX 460,67950
697-2175
42 M 74811 66 1M
1WAY, OLIVIA N, BOX 460 - 411 SUNSET,67950
697-2175
43 F 74811 68 P
PERIDO, DOMINADOR T, BOX 997,67950
697-2155
44 M 74801 68 GS

ELLINWOOD—316
(Barton County Society)

LAW, FINDLEY, MEDICAL ARTS BLDG,67526
564-2170
22 M 1902 51 FP
SHAPIRO, MARTIN, MEDICAL ARTS BLDG,67526
564-2170
54 M 74809 81 GP

ELLIS—913
(Central Kansas Society)

ROCHANAYCN, PIRA, 1204 WASHINGTON,67637
726-4956
43 M 89101 69 FP
SURFACE, GARDNER A, 101 WEST 13TH,67637
726-4454
02 M 1902 29 OD

ELLSWORTH—913
(Central Kansas Society)

DAVIS, GEORGE R, 308 KINGSLEY,67439
472-3121
19 M 1902 44 GS
O'DONNELL, HAROLD F, 412 BLAKE,67439
-
99 M 1902 26 OO
SEITZ JR, JOSEPH E, 308 KINGSLEY,67439
472-3121
22 M 1902 46 FP

EMPORIA—316
(Flint Hills Society)

AMEND, DOUGLAS J, 827 COMMERCIAL,66801
343-6565
46 M 1902 79 OBG
AUSTENFELD, MARK S, 2001 SCHAEFFER WAY,66801
-
53 M 1902
BARNETT, JAMES A, 919 W 12TH,66801
342-2521
54 M 1902 79 IM
BOSILJEVAC JR, JOSEPH E, 2522 W 15TH,66801
343-7043
51 M 1902 75 TS
BRADLEY, H RUSSELL, 1601 STATE,66801
343-2900
35 M 1902 61 FP
BROCKHOUSE, JOHN P, 1601 STATE,66801
343-2900
31 M 1902 57 IM
BURGESON, FRANK G, 1601 STATE,66801
342-6989
40 M 3005 65 DPH
BUTCHER, THOMAS P, 1128 LAWRENCE,66801
342-0722
05 M 1601 34 GS
CAMPBELL, EDWARD G, 1601 STATE,66801
343-2900
31 M 1902 61 FP
COLDSMITH, DONALD C, 1024 W 12TH,66801
342-7047
26 M 1902 58 FP
DAVIS, DAVID R, 1025 STATE ST APT #2,66801
-
02 M 2101 28 OO
EDWARDS, DAVID J, 1601 STATE ST,66801
343-1191
43 M 2803 69 ORS
ELLIS, BOBBY J, 1601 STATE,66801
343-2900
51 M 1902 77 1M
FARRIS, RONNIE S, 1601 STATE,66801
343-2900
50 M 3840 75 GS
FRAZIER, RICHARD L, 100S W 12TH,66801
342-5137
40 M 1902 72 GS
GANN, E LAMONTE, RR #2,66801
-
07 M 2802 37 OD
GARCIA, GOULD C, 919 WEST 12TH AVE,66801
342-2521
32 M 3607 58 1M
GEITZ, JAMES M, 919 W 12TH,66801
342-2521
46 M 1902 72 1M
GINAVAN, DUANE A, 1024 W 12TH,66801
342-5876
35 M 1902 62 FP
GLENN, JAMES N, 1601 STATE,66801
343-1191
40 M 4804 66 ORS
HARVEY, JOHN E, 2506 W 15TH,66801
343-2900
39 M 1902 65 OBG
HAVENHILL II, MARSHALL A, 812 ANDERSON ST,66801
343-6400
35 M 1902 61 OBG
HOPPER, CHARLES R, 25 W 5TH,66801
342-2341
17 M 1902 47 FP
HOWELL, BARBARA JOYCE, 2S10 W 15TH,66801
343-7590
45 F 3401 78 PD
KELLING, COLLYER, 1614 E WILMAN,66801
343-7676
50 M 1902 75 R
KNECHT, STEPHEN M, NEWMAN MEMORIAL HOSP,66801
342-7722
44 M 1902 70 R
KRETSINGER DO, W BRDCK, 1601 STATE,66801
343-2393
48 M 2878 77 1M
LUEGER, JAMES JOHN, D.D., NEWMAN MEM HDS,66801
343-6800
18 M 5605 43 PATH
MIGUELINO, OLIVER M, NEWMAN HOSP,66801
343-6800
35 M 74801 57 PATH
MORGAN, JOHN L, 919 WEST 12TH,66801
342-2521
15 M 4101 40 IM
NEIGHBOR, RALPH M, 827 COMMERCIAL ST,66801
343-6565
46 M 1902 72 OBG
NEUER, FREDERICK S, NEWMAN HOSP,66801
342-7722
46 M 3601 71 R
PARKER, WAYNE G, EMPORIA STATE UNIVERSITY,66801
343-1200
27 M 1902 56 FP
PASTOR, VICTOR HUGO, 1601 STATE,66801
342-7715
43 M 13202 68 U
RYAN, W SCOTT, 2S10 W FIFTEENTH,66801
343-7590
47 M 1902 73 PD
SAUL, F WILLIAM, ROUTE #5,66801
-
07 M 4113 40 OO
SCHELLINGER, RICHARD P, 1128 LAWRENCE,66801
342-0722
22 M 3005 49 GS
SNOWPARGER, MARVIN D, 1601 STATE STREET,66801
343-2900
29 M 1902 55 FP
SPEARS, CHESTER A, 12TH & CHESTNUT,66801
343-6800
50 M 2834 76 PATH
TRIMBLE SR, DAVID P, 517 MERCHANT STREET,66801
342-2572
04 M 1902 32 DPH
UNDERWOOD, CHARLES C, 25 WEST 5TH,66801
342-2341
07 M 1902 32 IM
VANDER VELDE, STANLEY L, 1601 STATE,66801
343-2900
16 M 1902 43 GS
WRIGHT, KENDALL M, 1024 WEST 12TH,66801
343-2376
45 M 1902 71 FP

ERIE—316
(Southeast Kansas Society)

8RYAN, EMERY C, RT 2 PDX 171,66733
-
04 M 1902 32 OD
HANDSHY, STANLEY E, PO BOX 256,66733
244-3291
54 M 1902 79 FP

ESKRIDGE—913
(Flint Hills Society)

WALKER, WILLIAM H, 2ND & CEDAR,66423
-
10 M 2401 38 IM

EUDORA—913
(Douglas County Society)

HOLLADAY, KENNETH R, 101 WEST 10TH,66025
542-2345
34 M 1902 58 FP

EUREKA—316
(Butler-Greenwood Society)

CISKEY, WILLIAM J, PO BOX 310,67045
583-7401
47 M 1902 72 FP
SKAER, STANLEY ALLEN, 100 E 16TH,67045
583-7486
40 M 3901 65 GS

FORT SCOTT—316
(Bourbon County Society)

AKERS, GUY I, 710 W 8TH,66701
223-3100
20 M 1902 53 FP
ALDIS, HENRY, 710 W 8TH,66701
223-3100
13 M 1902 41 DBG
ALDIS, WILLIAM, 710 WEST 8TH,66701
223-3100
20 M 1902 44 GS
BASHAM, JAMES J, 702 MEADOW LANE,66701
-
14 M 1902 37 OD
BENAGE, JOHN F, 821 BURKE,66701
223-2200
32 M 1902 58 DBG
BRAUN, EDWARD W, 710 WEST 8TH,66701
223-3100
42 M 1902 68 U
BURKE, JAMES J, 710 W 8TH,66701
223-3100
35 M 2834 61 IM
CHOW, STANLEY Y, 821 BURKE,66701
223-2200
18 M 24222 39 R
DUNSHEE, CARLYLE M, 710 W 8TH,66701
223-3100
32 M 1902 57 GS
GETTLER, DEAN T, 710 WEST 8TH,66701
223-3100
31 M 1902 57 GS
GOOD, JAMES T, 821 BURKE,66701
223-2200
21 M 2802 45 PATH
GRIMALDI, GARY A, 710 W EIGHTH,66701
223-3100
49 M 1902 74 OBG
IRBY, ADDISON C, 416 S JUDSON,66701
-
05 M 1606 28 OD
IRBY, PRATT, 710 WEST 8TH,66701
223-3100
13 M 4705 36 U
MCCANN, PATRICK E, 710 WEST 8TH,66701
223-3100
28 M 1902 59 IM
MCKENNA, MICHAEL J, 323 S JUDSON SUITE 120,66701
223-3950
38 M 1902 64 FP
NELSON, T EUGENE, 710 W 8TH,66701
223-3100
41 M 1902 69 FP
NICHOLS, ROBERT R, 102 S JUDSON,66701
223-4100
50 M 2803 76 FM
PHELPS, DAVID WAYNE, 102 S JUDSON,66701
223-4100
51 M 1902 76 FP
REEVES, CHARLES S, 710 W 8TH,66701
223-3100
37 M 1902 63 IM
SPENCER, JOHN HAROLD, 710 W 8TH,66701
223-3100
47 M 1902 74 FP
WEDDLE, DOUGLAS P, 710 WEST 8TH,66701
223-3100
43 M 1720 69 FP

FREDONIA—316
(Southeast Kansas Society)

BACANI, OSWALDO, PO BOX 576,66736
378-3700
44 M 74810 70 GS
BAYLES, HUGH G, PO BOX 30,66736
378-3412
25 M 1902 52 FP
BEAL, RAYMOND J, 136 S FIFTH,66736
-
12 M 1902 38 OD
RINDT, PHILLIP L, 432 N SEVENTH,66736
378-2298
45 M 1902 71 FP
SUMNER, RALPH N, 712 MADISON,66736
378-2311
31 M 1902 57 FP

FT. LEAVENWORTH — 913
(Leavenworth County Society)

CHAVALA, SUDARSAN, EYE CLINIC,66027
684-4157
43 M 49511 68 DPH

GARDEN CITY—316
(Southwest Kansas Society)

ARROYO, ZEFERINO, 2124 ANTLER RIDGE DR,67846
872-2187
M 74802 GS
AUSTIN, JOHN D, 601 N 6TH,67846
276-2346
14 M 1601 40 FP
BARNARD III, JAMES A, 1601 HARDING,67846
275-9671
31 M 4812 62 DBG
BEGGS, DAVID F, BOX 1077,67846
275-9671
39 M 1902 64 IM
BIGLER, F CALVIN, 801 N FIFTH,67846
275-2141
31 M 801 57 GS
BRUND, JAMES W, 1133 KANSAS PLAZA,67846
276-8201
42 M 4706 66 FP
CALBECK, JOHN, 2603 BELMONT PLACE,67846
275-9671
50 M 1902 75 IM
COLLINS, SHARON A, 603 N FIFTH,67846
275-9671
51 F 2512 78 PD
EICHHORN, FRANK D, BOX 719,67846
276-8132
25 M 1902 56 FP
FENTON, ROBERT M, 603 N FIFTH,67846
275-9671
20 M 1902 54 FP
FRY, LUTHER L, ST CATHERINE HOSP,67846
275-7248
41 M 1902 67 DPH
GARDINER, TED M, 603 N FIFTH,67846
275-9671
48 M 506 74 PD
GILBERT II, JOHN H, BOX 1077,67846
275-9671
46 M 1902 70 DRS

GREENWOOD, JAMES F, PO BOX 419,67846
356-1261
33 M 1611 65 FP
HANSEN, FRANK W, 603 FIFTH,67846
275-9671
49 M 1902 76 PM
HUNSBERGER, TERRY R, D.O., 602 N THIRD,67846
275-7128
47 M 2878 73 FP
JACKSON, MICHAEL D, 605 N FIFTH,67846
275-9671
51 M 4814 76 FP
KALBAC, RICHARD W, BOX 1077,67846
275-9671
45 M 2803 70 OBG
KOKSAL, TOM, PLAZA MED CENTER,67846
276-8201
M 1902 FP
MELIN, BRUCE D, 608 N FIFTH,67846
275-6111
51 M 5605 77 PATH
MEYERS, STEPHEN, BOX 1077,67846
275-9671
48 M 2834 70 PO
MILLER, ROBERT E, BOX 1077,67846
275-9671
26 M 4812 55 GS
RODRIGUEZ, PAUL L, BOX 1729,67846
275-6111
39 M 4706 66 R
SHUSS, JOHN L, 801 N FIFTH,67846
275-2141
49 M 1902 75 GS
SPIKES, MARION E, 603 N 5TH,67846
275-9444
26 M 1902 62 FP
TEARE, MAX E, 1007 DAVIS,67846
276-7689
28 M 1902 54 P
TURNER, JOHN W, 210 E SPRUCE,67846
276-3292
13 M 1902 39 FP
VACHAL, EVA, 608 N FIFTH,67846
275-6111
F 1902 PATH
VAN ANTWERP, JAMES, 306 CEDAR SUITE 201,67846
275-5141
30 M 1902 55 P
WILEY, HORACE M. BOX 1136, 67846
276-6901
12 M 2802 40 GS
ZELLER, MYRON J, BOX 1077,67846
275-9671
38 M 1902 64 OM

GARDEN PLAIN—316
(Sedgwick County Society)

LIND II, EDWARD J, 728 BIERMANN,67050
535-2218
53 M 1902 78 FP
REINHARDT-WULF, TAISSIA L, PO BOX 273,67050
-
19 F 91302 42 OO

GARDNER—913
(Johnson County Society)

NIKNIA, SEYED M, 427 W MAIN,66020
884-8711
38 M 51701 67 GS
REECE, A THOMAS, 427 W MAIN,66030
884-8711
37 M 1902 63 FP
WHITAKER, MARK A, 136 E MAIN,66030
884-7822
53 M 1902 77 PO

GARNETT—913
(Anderson County Society)

DOUGHERTY, THOMAS M, 117 W 6TH,66032
448-5421
28 M 1902 55 FP
HARRIS JR, CLAIB B, 320 S OAK ST,66032
448-5431
17 M 1902 44 FP
HENDERSON, DAVID V, 117 W SIXTH,66032
448-5421
48 M 1902 79 FP
LEITCH, DAVID A, GARNETT MEDICAL CENTER,66032
448-5421
38 M 1902 63 FP
STEVENS, MILORED J, 202 W 4TH,66032
448-5454
23 F 1902 47 FP
STEVENS, ROBERT L, 202 WEST 4TH,66032
448-5454
23 M 1902 47 FP

GIRARD—316
(Crawford County Society)

FRIGGERI, ROBERT W, 111 N SUMMIT,66743
724-8723
23 M 1902 51 FP
HALL, WESLEY H, PO BOX 158,66743
724-6154
25 M 1902 57 FP
WILKINS, JAMES T, BOX 158,66743
724-6154
51 M FP

GLASCO—913
(Cloud County Society)

HARWOOD, CLAUDE J, PO BOX 428,67445
568-2245
25 M 1902 55 FP

GOODLAND—913
(Northwest Kansas Society)

AUSTIN, KENNETH O, 520 MAIN,67735
899-5651
33 M 3005 63 FP
FITZGERALD, E JAMES, BOX 871,67735
899-5695
44 M 74811 77 GP
LONG, LLOYD O, 520 MAIN,67735
899-5651
37 M 1720 63 FP
MCCULLOUGH, ROBERT C, 126 E 10TH BOX 180,67735
899-3633
25 M 702 58 GP
OLSON, CLITUS W, 520 MAIN ST,67735
899-5651
16 M 3005 48 GS

GREAT BEND—316
(Barton County Society)

ALDERSON, THOMAS W, 3520 LAKIN,67530
792-5341
50 M 1902 75 FP
BEAHM, ANOL W, 3923 BROADWAY,67530
793-7827
16 M 1902 43 FP
BEAHM, DONALD E, MED ARTS BLDG,67530
792-3626
45 M 1902 71 OPH
CAVANAUGH, CLAIR J, C K M C,67530
792-2617
23 M 1803 47 R
DEGNER, JAMES R, 3515 BROADWAY,67530
792-2617
31 M 1902 57 R
ELLEDGE, E FRED, 2222 FOREST,67530
792-3678
38 M 1902 65 U
EVANS, WILLIAM R, 1912 LINCOLN,67530
-
25 M 1902 53 OO
FIESER, CARL W, 3515 BROADWAY,67530
792-2617
45 M 1902 71 R
FLESKE, LEONARD T, 1315 POLK,67530
792-4383
49 M 1902 75 ORS

GATEND, JOSEPH, 1023 JACKSON SQUARE,67530
793-3501
25 M 64901 50 OBG
HILL, LARY MICHAEL, 1017 A JACKSON,67530
793-8141
51 M 1902 77 FP
HOLT, JOHN M, PO BOX 1328,67530
793-8429
35 M 1902 61 IM
JONES, EDWARD L, 3515 BROADWAY,67530
792-2511
35 M 1902 61 PATH
KING, WILLIAM T, 3421 FOREST,67530
793-3501
35 M 1902 61 OBG
KIRBY, MERLIN G, 3520 LAKIN,67530
793-3091
31 M 1902 56 GS
KRUEGER, HAVEN C, 1023 JACKSON SQUARE,67530
792-2163
32 M 1902 61 PD
MCALLASTER, WENDALE E, 2111 FOREST,67530
793-3591
24 M 1902 54 GS
NIEDEREE, W CURTIS, 3520 LAKIN,67530
793-3091
30 M 3006 56 GS
POLSON, ROBERT C, BOX A 1422 POLK ST,67530
793-8414
17 M 1902 42 DPH
PRESTON, RICHARD, PO BOX 1328,67530
793-8426
42 M 1902 69 IM
REPLOGLE, CHARLES B, 2111 FOREST,67530
793-3591
27 M 1902 53 FP
RUIZ, CARLOS M, PO BOX 1348,67530
792-3210
25 M 27501 52 P
SAYLER, JEROME, CENTRAL KS MEDICAL CENTER,67530
792-2511
20 M 4113 50 PATH
SCHUETZ, PERRY N, 1422 POLK BOX A,67530
793-8414
45 M 1902 71 OPH
SCHUKMAN, JAY S, 3520 LAKIN,67530
792-5341
50 M 1902 75 FP
SHIVEL, DAVID G, 3523 FOREST,67530
793-3523
28 M 1902 55 FP
SMITH, PERRY MILTON, 3520 LAKIN,67530
792-5341
52 M 1902 77 FP
SWAN, MAJOR MARTIN, 3923 BROADWAY,67530
792-4540
06 M 1902 43 IM
UNREIN, ROBERT J, 1017A JACKSON,67530
792-2504
29 M 1902 58 FP
WHITE, CHARLES L, 2412 DOVE TERRACE,67530
-
06 M 1902 36 DD
WIGGS, JAMES W, 1027 JACKSON,67530
792-1336
36 M 1720 63 N
WIKOFF, DONALD L, 3520 LAKIN SUITE 108,67530
792-7353
49 M 3005 75 U
YOUN, HWAN, 3515 BROADWAY,67530
792-2617
48 M 58310 73 DR

GREENSBURG—316
(Iroquois County Society)

BRADLEY, J RODERICK, BRADLEY-WALDORF CLINIC,67054
723-2127
23 M 1902 47 FP
WALDORF JR, MELVIN H, BRADLEY-WALDORF CLINIC,67054
723-2127
23 M 1902 47 FP

HALSTEAD—316
(Harvey County Society)

AILLON, ALEJANDRO J, HERTZLER CLINIC,67056
835-2241
39 M 26402 63 TS
BAILEY, COLIN, HERTZLER CLINIC,67056
835-2241
33 M 35205 59 GYN
BEUGELSDIJK, HENRY PETER, 421 SPRUCE,67056
835-2241
49 M 1902 74 ANES
BURNETT, A DEAN, HERTZLER CLINIC,67056
835-2241
21 M 1902 52 GS
DECKER, DONALD D, HERTZLER CLINIC,67056
835-2241
31 M 1902 56 CD
EASTES, GARY DEAN, HERTZLER CLINIC,67056
835-2241
44 M 4812 71 U
GNAU, FREDRIC B, 803 MAIN,67056
835-2241
42 M 1902 68 DTO
HAIRE, WILLIAM D, 901 MAIN,67056
835-2241
49 M 1902 74 HEM
HARMS, WILMER A, THE HERTZLER CLINIC,67056
835-2241
22 M 1902 56 DPH
HOOFER, WILFORD D, HERTZLER CLINIC,67056
835-2241
30 M 1902 55 TS
KIMMEL, KENNETH K, 4TH & CHESTNUT,67056
835-2241
52 M 1902 77 IM
MALONE, EUGENE M, HERTZLER CLINIC,67056
835-2241
23 M 1902 56 IM
MARSH, CONNIE M, HERTZLER CLINIC,67056
835-2241
47 F 1902 75 IM
MONTGOMERY, LLOYD DAN, HERTZLER CLINIC,67056
835-2241
43 M 3601 69 P
MONTGOMERYSHORT, RUTH G, HERTZLER CLINIC,67056
-
10 F 1902 37 ENT
MORTON, JOHN E, 320 WALNUT,67056
-
99 M 35211 26 DO
RESCHLY, RON, 603 CHESTNUT,67056
835-2241
M 3806 ORS
RIZZA, ROBERT G, RT #2,67056
-
30 M 1201 56 PD
SHAH, SHARFUDDIN, HERTZLER CLINIC,67056
835-2241
31 M 70401 58 IM
STOFFER, ROBERT P, HERTZLER CLINIC,67056
835-2241
26 M 1902 48 IM
TEJANO, NEONILO A, HERTZLER CLINIC,67056
835-2241
43 M 74808 67 ORS
WELCH, JACK W, HERTZLER CLINIC,67056
-
18 M 1902 51 DD
WILSON, H RANDOLPH, HERTZLER CLINIC,67056
835-2241
20 M 4112 45 GYN

HANOVER—913
(Northeast Kansas Society)

WARREN, LINDA D, BOX 38,66945
325-2240
44 F 1902 70 FP
WARREN, ROGER D, BOX 38,66945
337-2214
31 M 1902 57 GS

HARPER—316
(Tri-County Society)

BELLAR, RALPH E, 1019 CENTRAL,67058
896-3131
31 M 3005 60 FP
FORRED JR, WALTER A, 121 E MAIN,67058
896-3661
43 M 1902 69 FP
GARDNER, BILLIE L, 121 E MAIN,67058
896-3661
25 M 1902 57 FP

HAYS—913
(Central Kansas Society)

*ALBERS, ROBERT C, 2707 VINE,67601
-
48 M
ALLEN, JAMES E, 2707 VINE #6,67601
628-3261
46 M 1902 72 1M
APPLEGATE JR, FRANCIS R, 1010 DOWNING,67601
628-8218
30 M 1902 55 OPH
ARTMAN, JOHN C, 2005 LINCOLN DRIVE,67601
625-2518
24 M 2002 48 ANES
BULA, RALPH E, 3209A WILLOW,67601
-
12 M 1902 37 OO
CARLSON, EARL V, DRAWER 430,67601
628-8221
31 M 3005 56 ORS
CECIL III, JOHN, BOX 833,67601
625-6521
43 M 4804 69 R
COOY, DOROTHY, 2704 WOODROW CT,67601
628-4000
29 F 3607 53 P
COOY, JOHN, 2704 WOODROW CT,67601
628-2871
25 M 401 60 P
COX, ROBERT H, 2507 CANTERBURY RD,67601
628-3051
43 M 1902 70 PD
CRAMM, RUSSELL E, 3004 BROADWAY,67601
625-2518
30 M 1902 56 GS
DORSCH, JOHN N, 2508 CANTURBURY,67601
628-6151
54 M 1902 79 FP
DYCK, ERIC LEE, FAMILY PRACTICE CLINIC,67601
628-6151
52 M 1902 77 FP
EDDY, VICTOR M, 105 W 13TH,67601
625-2551
29 M 1902 55 GS
ENBERG, ROBERT, 2507 CANTERBURY,67601
628-3051
43 M 1902 69 PD
HAIGLER, JAMES P, 234 WEST 11TH,67601
625-2537
13 M 3006 39 GP
HALLING, L WILLIAM, 1300 EAST 13TH,67601
625-5646
27 M 5002 57 PATH
HANSA, HANSA, 2503 CANTERBURY,67601
628-3245
45 M 89101 69 OBG
HUTCHISON, GLEN C, 3200 COUNTRY LANE,67601
628-8251
21 M 1902 50 ANES
HUTCHISON, MARC K, 213 CASTILLIAN GARDENS,67601
-
M
KANE JR, WILLIAM M, CANTERBURYS CLINIC PA,67601
628-3245
27 M 1001 54 OBG
KELLY, A CHRISTINE, 2707 VINE SUITE 7,67601
628-3217
49 F 2846 77 GS
KIFER, C JAMES, BOX 833,67601
625-6521
45 M 1902 71 OR
LASLEY, MICHAEL B, 2707 VINE SUITE 7,67601
628-3217
45 M 1902 71 GS
LOEB, ELBIE L, 2209 CANTERBURY RD,67601
625-4224
51 M
MATTICK, IRVIN H, 2818 N VINE,67601
628-8221
18 M 2802 43 ORS
MYRICK, MICKEY, 2501 CANTERBURY RD,67601
628-6151
42 M 3005 74 FP
NEIL, ROY NEWTON, 1108 MAIN ST S 2018,67601
628-3215
38 M 3005 65 PATH
NEWCOMB, WARD M, 1300 E 13TH,67601
625-5646
47 M 3005 71 PATH
NIELSEN, MARY L, 1300 E THIRTENTH,67601
625-5646
47 F 1902 77 PATH
RAJEWSKI, RICHARD L, 2509 CANTERBURY,67601
628-6151
51 M 1902 76 FP
REYNOLDS, JEFFREY C, 2517 CANTERBURY RD,67601
625-7311
39 M 1902 64 ENT
REYNOLDS, LLOYD W, 504 W 36TH,67601
-
10 M 3840 34 OO
RICHARDS, DALLAS LEE, 2209 CANTERBURY RD STE A,67601
625-4224
49 M 1902 76 1M
ROBINSON, MICHAEL D, 2209 CANTERBURY RD,67601
625-4224
50 M 3005 76 1M
RUTNGAMLUG, LUECHA, 3004 BROADWAY,67601
628-6175
40 M 89101 68 GS
SILER, EUGENE T, 1010 DOWNING,67601
628-8218
24 M 1902 52 OPH
STADALMAN, ROSS EUGENE, 2707 VINE SUITE 7,67601
628-3217
47 M 1902 73 GS
WATTS, HARRY E, 1010 DOWNING AVE,67601
628-8218
27 M 702 54 OPH
WEBER, WALLACE N, 2707 VINE SUITE 10,67601
628-3231
43 M 1902 69 O
WERTH, DARRELL DEAN, 2209 CANTERBURY RD STE D,67601
628-6014
50 M 1902 75 U
WIEGMAN, HUGH ALAN, BOX 833,67601
625-6521
34 M 1803 60 R
WILCOX JR, HOWARD L, PO DRAWER 430,67601
628-8221
44 M 1902 70 ORS

HAYSVILLE—316
(Sedgwick County Society)

MORGAN, NOVA L, 301 W GRAND,67060
225-1371
20 M 3901 50 FP
SAYEED, BASEER A, 138 STEWART,67060
529-0450
49 M 49504 74 1M

HERINGTON—913
(Dickinson County Society)

BUSTOS, JONAS G, 1005 NORTH B,67449
258-3705
41 M 74810 68 GS
DOZIER, FRED S, 1005 NORTH B STREET,67449
258-2215
10 M 4804 34 FP

HESSTON—316
(Harvey County Society)

DIENER, CLAYTON H, PO BOX 386,67062
327-4122
18 M 1902 54 GS
FRIESEN, FLORENCE V, SHOWALTER VILLA,67062
-
87 F 1611 14 OO
YODER, VERNON E, ROUTE #1,67062
283-2400
31 M 4802 61 P

HIAWATHA—913
(Northeast Kansas Society)

BASSETT, P MARCUS, 202 S SIXTH,66434
742-7915
51 M 1902 77 FP
DUCKETT, THOMAS G, 201 MIAMI,66434
-
10 M 1902 34 OO
LARSON, DELBERT L, 314 OREGON,66434
742-2161
30 M 1803 64 FP

MEIDINGER, RAY, 111 S FOURTH,66434
742-2135
03 M 3005 32 FP
SINNING, GARY, 314 OREGON,66434
742-2161
49 M 1902 74 FP

HIGHLAND—913
(Northeast Kansas Society)

HALL JR, ERNEST B, BOX 96,66035
442-3341
53 M 1902 78 FP

HILL CITY—913
(Central Kansas Society)

REDDY, B N, 412 N 12TH,67642
674-2255
38 M 49557 66 PO
REDDY, P JAGANNADHA, 80 WALNUT DRIVE,67642
674-2255
42 M 49511 66 GS

HILLSBORO—316
(Marion County Society)

ENS, GERHARD GEORGE, 613 SOUTH MAIN,67063
947-5931
20 M 1902 55 FP
ENS, PETER, 209 SOUTH MAIN,67063
947-3671
14 M 1902 51 FP
FRANZ, ROBERT G, 704 S MAIN,67063
947-3197
33 M 3901 59 FP
LOEWEN, PETER S, SKILLED NURSING UNIT,67063
91 M 1902 30 DO

HOISINGTON—316
(Barton County Society)

FERNANDEZ, HECTOR O, 351 W TENTH,67544
653-4944
41 M 74809 66 GS
HOOPER, ALLEN, 353 W TENTH,67544
653-4138
50 M 1902 79 FP
MOORE, ROBERT, 814 NORTH ELM,67544
653-2151
22 M 3901 53 FP

HOLTON—913
(Jackson County Society)

MOSER, ERNEST C, 438 HILLCREST DR,66436
09 M 1902 34 DO
MOSER, M ROSS, 418 WEST FIFTH,66436
364-3116
19 M 1902 47 FP
MOSER, ROY H, 801 IOWA,66436
04 M 1902 32 DO
SEELEY, JAMES C, 418 WEST 5TH,66436
364-3116
34 M 1902 64 FP

HORTON—913
(Northeast Kansas Society)

FRANCISCO, EDGARDO, PO BOX 6,66439
486-2646
39 M 74802 57 GP
WALTON, PHILIP O, 300 W 18TH,66439
486-2828
32 M 1902 63 FP

HOXIE—913
(Northwest Kansas Society)

NEUENSCHWANDER, JOHN, 1401 QUEEN,67740
675-3292
26 M 2802 51 FP
NEUENSCHWANDER, JOHN RAND, 700 MAIN,67740
675-3292
47 M 1902 72 FP

HUGOTON—316
(Seward County Society)

FREDERICK, M F, 1006 S JACKSON,67951
544-2784
20 M 1902 44 FP
IHRIG, ROGER W, 1006 S JACKSON,67951
544-2784
49 M 1902 74 FP
LENEVE, ROBERT T, 1006 S JACKSON,67951
21 M 3901 46 DO

HUMBOLDT—316
(Southeast Kansas Society)

LONG, EDWARD E, 8TH & NEW YORK,66748
473-2411
21 M 1902 50 FP

HUTCHINSON—316
(Reno County Society)

ADAMS JR, MARCUS W, 2101 N WALDRON,67501
663-6121
33 M 3901 59 PO
ALBRIGHT, JEROLD O, 2101 N WALDRON,67501
663-6121
39 M 1902 66 FP
BARKER, STANTON L, 2101 N WALDRON,67501
663-6121
54 M 1902 79 FP
BAUER, THOMAS A, 2101 N WALDRON,67501
663-6121
41 M 1902 67 1M
BLANK, JOHN N, ROUTE 5 BOX 220,67501
07 M 1902 38 DO
BORRA, MARIO J, 1 WEST 8TH,67501
662-1751
24 M 2401 47 U
BOS, NORMAN C, 2101 N WALDRON,67501
663-6121
24 M 1611 47 ORS
BOUDA, DAVID W, 2101 WALDRON,67501
663-6121
45 M 1902 72 1M
BRADA, DONALD ROBERT, 308 W 20TH,67501
662-6041
39 M 1902 65 P
BURGER, J DALE, 2101 N WALDRON,67501
663-6121
21 M 1902 46 FP
CASEY, JAMES, 2101 N WALDRON,67501
663-6121
42 M 3005 69 PO
CHERVEN, PHILIP L, 1100 N MAIN,67501
662-3364
45 M 2501 71 PO
DEPENBUSCH, FRANCIS L, 1708 E 23RD,67501
663-7187
38 M 1902 65 DPH
DUHON, JOHN MARQUE, 1701 E 23RD,67501
665-2126
47 M 4802 75 OR
ECKART, DE MERLE E, 201 N MAIN #315A,67501
662-4921
14 M 1902 40 P
FALTER, RICHARD T, 1708 E 23RD ST,67501
663-7187
38 M 1902 67 OPH
FOSS, DANIEL C, 2101 N WALDRON,67501
663-6121
43 M 1902 69 GE
GOODPASTURE, WILLARD C, RR 3 BOX 45,67501
10 M 1602 36 DO

GRAVES, KATHRYN, 2101 N WALORON,67501
663-6121
49 F 1902 74 O
HEDRICK, KENNETH E, 2101 N WALORON,67501
663-6121
27 M 1902 53 GS
HOLDERMAN, WALLACE O, 2101 N WALORON,67501
663-4406
28 M 1902 54 ORS
JARROTT, JOHN B, 1100 N MAIN,67501
663-2151
16 M 1902 40 ORS
JOHNSON, RICHARD L, 2101 N WALORON,67501
663-6121
M R
KEPKA MD, DENNIS J, 3117 S MEADOWLAKE DR,67501
663-2151
43 M 56101 77 FP
KLOSTERHOFF, BRUCE E, 1701 E 23RD,67501
663-2108
45 M 1611 71 P
LESSER, DANE A, 2101 N WALORON,67501
663-6121
49 M 3901 75 U
LETTNER, HANS T, BOX 159,67501
662-7801
23 M 40716 49 PATH
LUKENS, DAVID, 2101 N WALORON,67501
663-1136
24 M 2307 48 IM
MARTINEZ, JOHN D, 1100 N MAIN,67501
663-2151
48 M 1902 74 IM
MCCOY, CHARLES T, 310 WOLCOTT BLDG,67501
662-0121
16 M 1902 41 OPH
MCMULLEN, JOSEPH E, 2101 N WALORON,67501
663-6121
33 M 1902 62 GS
MULL, JOHN C, 2101 N WALORON,67501
663-6121
34 M 1902 61 OBG
NEFL, WILBUR B, 2101 N WALORON,67501
663-6121
24 M 1902 59 IM
NEUSCHAFER, DARREL R, 2101 N WALORON,67501
663-6121
48 M 1902 74 OBG
NUNEMAKER, MARION E, PO BOX 1129,67501
662-5391
21 M 1902 46 ANES
OPENSHAW, CALVIN R, 2020 N WALORON,67501
662-0569
21 M 4901 44 GS
PAINE, GEORGE E, 220 W 23RD,67501
-
94 M 1606 19 OO
PEASE, GARY L, 1712 E 23RD,67501
662-4458
41 M 3005 67 OTO
PERKINS, JACK L, 2101 N WALORON,67501
663-6121
24 M 1902 53 FP
RATE, PEGGY S, 2221 E 56TH,67501
663-6121
46 F 1902 73 PO
RATE, ROBERT R, 2221 E 56TH,67501
663-6121
47 M 1902 74 IM
SCALES, WILLIAM M, PLAZA TOWERS,67501
-
00 M 1803 31 OO
SCHEEL, BRADLEY J, 1100 N MAIN,67501
663-2151
48 M 1902 74 IM
SCHROLL, JACK C, 2101 N WALORON,67501
663-6121
24 M 1902 49 OBG
SHAW JR, JAMES W, BOX 1646,67501
662-7801
40 M 1902 65 PATH
SHEARS, ROBERT N, 1100 N MAIN,67501
662-3364
20 M 1902 44 PO
SMITH, THOMAS WILLIAM, 1712 E 23RD,67501
662-4458
43 M 1643 68 OTO
SPITZER, JEROME S, 1100 N MAIN,67501
663-2151
33 M 3005 59 FP
STAFFORD, ROBERT W, 2101 N WALORON,67501
663-6121
43 M 2101 69 IM

STENSAAS, CARL O, 401 WOLCOTT BLDG,67501
663-9147
10 M 1902 38 O
STOUT, JAMES M, 2101 N WALORON,67501
663-6121
29 M 1902 55 FP
SUMNER, JOYCE R, 3011 - B NUTMEG LANE,67501
662-0822
26 F 1902 51 ANES
SUMNER, MARION M, 3011-B NUTMEG LN,67501
662-0822
26 M 1902 52 IM
TAYLOR, E J, 1100 N MAIN,67501
663-2151
34 M 1902 61 FP
TISDALE, TERRANCE C, HUTCHINSON CLINIC PA,67501
663-6121
36 M 6701 61 ORS
VON RUDEN, WILLIAM J, 2100 N WALORON,67501
662-3306
26 M 1611 52 GS
WEIDENSAUL, D N, 2101 N WALORON,67501
663-6121
50 M 1902 75 IM
WILLIAMSON, JOHN, 1100 N MAIN,67501
669-0191
35 M 2501 60 ORS
WORTMAN, JACK A, 2101 N WALORON,67501
663-6121
34 M 1902 62 IM

INDEPENDENCE—316
(Southeast Kansas Society)

BAIR, ALBERT E, PO BOX 925,67301
331-7650
16 M 1902 44 GS
BARBERA, PORTER E, 800 WEST CHESTNUT,67301
331-4400
19 M 4707 46 FP
BEAHM, EDGAR H, 307 PROFESSIONAL BLDG,67301
331-2311
13 M 1902 44 FP
CHAPPUIE, WILLIAM G, 800 CHESTNUT WEST,67301
331-5440
24 M 1902 51 FP
EMPSON, CHARLES L, 400 N 14TH,67301
331-6019
37 M 1902 68 FP
KNUTH, KENNETH L, 2900 TERRA VISTA,67301
331-2200
22 M 1902 50 R
MYERS JR, EARL B, BOX 548,67301
331-3420
32 M 2803 64 GS
OSBORN, ROBERT M, O.D., PO BOX 826,67301
331-2070
M 2843 69 GP
PATEL, V C, PO BOX 883,67301
331-2725
50 M 49576 74 ORS
PHIPPS, RONNY G, PO BOX 843, 67301
331-7901
54 M 512 FP
PRENTICE, WALTER B, PO BOX 628,67301
331-8650
29 M 1643 56 OR
ROBINSON, EDGAR L, 209 NORTH 6TH,67301
331-1450
15 M 1902 42 FP
STACEY, KIMBALL, 800 W CHESTNUT,67301
331-6350
M IM
SUTTON, ROBERT E, PO BOX 1003,67301
331-7700
46 M 1902 72 FP
TONG, ROLANDO M, PO BOX 425,67301
331-6573
49 M 74801 72 GS

IOLA—316
(Allen County Society)

COPENING, TELL B, 305 N WASHINGTON,66749
365-2134
43 M 1902 69 FP
DETAR, GEORGE F, 219 W MADISON,66749
365-5174
28 M 1902 57 GS

OICK, WILLIS G, BOX 826,66749
365-2131
13 M 512 41 GS
HANSON, OAVID C, 501 E MADISON BOX 714,66749
365-2131
46 M 512 73 FP
LENSKI JR, FRANCIS X, 206 S JEFFERSON,66749
365-3901
26 M 1606 49 FP
MYERS, W EUGENE, 211 S STREET,66749
365-3732
12 M 1902 46 FP
PEES, GERALO B, 219 W MADISON,66749
365-5175
15 M 1902 43 GS
SCHMAUS, LYLE F, 1020 N JEFFERSON,66749
-
99 M 1803 26 OO
WOLFE, BRIAN O, 305 N WASHINGTON,66749
365-2134
M FP

JETMORE—316
(Ford County Society)

O'SHEA, JAMES G, BOX 545,67854
357-8321
18 M 3901 48 FP

JEWELL—913
(Mitchell County Society)

PLOWMAN, CARL W, ,66949
-
99 M 1606 26 FP

JOHNSON—316
(Southeast Kansas Society)

OAILEY, RONALO F, BOX 470,67855
492-6230
31 M 1902 57 FP

JUNCTION CITY—913
(Geary County Society)

BOLLMAN, CHARLES S, PO BOX 397,66441
762-4575
41 M 3901 66 GS
BRETHOUR, LESLIE J, 1106 ST MARYS RO,66441
238-4151
13 M 3006 39 FP
BUNKER JR, HERBERT L, 1106 ST MARYS RO,66441
238-5131
20 M 1606 45 FP
COPELANO, GARY A, GEARY COMMUNITY HOSP,66441
762-2387
42 M 1902 68 R
CRAIG, THOMAS A, 1106 ST MARYS RD STE 102,66441
762-4255
53 M 1902 78 1M
FIELDSTONE, PAUL R, 1106 ST MARYS RD STE 204,66441
762-4300
46 M 6501 71 U
LABHSETWAR, S A, MEOICAL ARTS BLOG,66441
762-4147
39 F 49528 64 OBG
MACE, RONALO O, PO BOX 163,66441
762-4884
42 M 3901 74 FP
MINNICK, CHARLES V, 435 W CHESTNUT,66441

08 M 2105 35 FP
O'OONNELL, HARRY E, 1106 ST MARYS RO,66441
762-4947
14 M 4113 42 FP
PATEL, MAHENDRA N, 1106 ST MARYS RD STE 206,66441
762-2327
48 M 35207 74 1M
SCOTT, ALEX, 507 WEST 6TH,66441
238-2518
23 M 5605 48 FP
WRIGHT, STANLEY E, 1106 ST MARYS RO,66441
762-4884
47 M 3901 74 FP

KANSAS CITY, KANSAS—913
(Wyandotte County Society)

ABOOU, NABIH I, KU MEOICAL CENTER,66103
588-6586
34 M 33002 58 A
ALEXANOER, CHARLES E, TWO GATEWAY CENTER #917,66101
321-6670
43 M 401 70 OBG
ALEXANOER, CLYOE W, 1514 NORTH 5TH,66101
281-4380
96 M 4707 23 FP
ALGIE, WILLIAM H, 907 N 7TH,66101
371-5079
02 M 1902 27 1M
ALLEGRE, ANN, 1225 N 78TH,66112
788-7099
50 F 1902 77 1M
ALLEN, TIMOTHY E, 9201 PARALLEL PKWY,66112
651-7195
49 M 1902 76 R
ALLEN, WILLIAM R, 9201 PARALLEL PARKWAY,66112
334-4110
22 M 1902 46 R
AMARE, MAMMO, KU MEOICAL CENTER,66103
588-6031
36 M 60501 61 1M
ARAKAWA, KASUMI, KU MEO CENTER,66103
588-6670
26 M 57249 53 ANES
ARENAL, ANGELA C, BETHANY HOSPITAL,66102
281-8400
38 F 35204 60 ANES
ASHER, MARC A, K U MED CENTER,66103
588-6130
36 M 1902 62 ORS
ATKINS JR, FLOYO L, K U MED CENTER,66103
588-6015
43 M 5104 69 CO
BAILIE, MICHAEL OAVID, KU MEO CENTER,66103
355-6339
36 M 1720 64 PNP
BASINGER, BRAOLEY B, 3824 BOOTH APT 3,66103
-
53 M 1902
BASS JR, LEWIS N, 1975 N 5TH,66101
321-2320
21 M 1902 45 PO
BATNITZKY, SOLOMON, K U MED CENTER,66103
588-6835
40 M 83601 64 OR
BECKER, LESLIE E, 600 NEBRASKA,66101
342-4010
23 M 1003 46 U
BEJAR, JOSE M, 3838 RAINBOW BLVO APT 609,66103
588-6970
46 M 31901 74
BENSON, KIRK T, KU MEO CENTER,66103
588-6670
54 M 1902 79 ANES
BERGIN, JAMES J, BETHANY MED CENTER,66102
281-8767
28 M 2407 54 IM
BICHLMEIER, FRANKLIN G, 155 S 18TH,66102
371-6800
33 M 1902 58 COS
BILLINGSLEY, THAO H, 155 S 18TH,66102
321-3844
41 M 1902 66 P
BIXLER II, THOMAS J, KU MEO CTR CAROIOVAS SURG,66103
588-6107
47 M 1205 74 COS
BOOENSTEINER, OAVIO C, K U MEO CENTER,66103
588-6077
51 M 1803 76 IM
BOGGAN, MICHAEL O, 155 S 18TH,66102
371-6800
40 M 1201 67 COS
BOLINGER, ROBERT E, KU MEOICAL CENTER,66103
588-6022
19 M 1902 43 END
BOLTON, VICTOR E, 9919 PARALLEL PARKWAY,66112
371-2020
24 M 1902 48 OBG
BOSILEVAC, FREO N, 155 S 18TH,66102
-
16 M 1902 44 OPH
BRACKETT JR, CHARLES E, KU MEO CENTER,66103
588-6117
20 M 3501 44 NS
BRILLHART, MAXINE T, 1610 WASHINGTON BOULEVARO,66102
321-4800
15 F 1902 50 FP

BROOKS, WILLIAM HENRY, 155 S 18TH,66102
371-4343
49 M 1902 74 R
BROWN, JOHN V, 3728 CAMBRIDGE,66103
432-5610
58 M 1902
BUBB, STEPHEN K, 155 S 18TH,66102
371-6802
48 M 1902 74 ORS
BURGER, WILLIAM E, 355 NEW BROTHERHOOD BLDG,66101
371-1017
21 M 3006 51 GS
BUTTERFIELD, MARI ANNE, KU MED CENTER,66103
588-6670
52 F 1902 77 ANES
BYERS, MARTHA S, 4201 ORVILLE,66102
596-1313
54 F 1902 79 FP
CALDERON, JAIME, 4631 ORVILLE SUITE 201,66102
596-1185
39 M 26401 66 IM
CAMERON, WILLIAM J, KU MEDICAL CENTER,66103
588-6200
29 M 2501 54 OBG
CARLIN, JAMES WILLARD, KU MED CENTER,66103
588-6670
51 M 1902 76 ANES
CARPENTER, PAUL R, 155 SOUTH 18TH,66102
371-6800
24 M 1902 50 TS
CARRO, TONY L, 4338 LLOYD, 66103
—
57 M 1902
CHALIAN, ALEXANDER R, 2648 MINNESOTA AVENUE,66102
03 M 3509 37 DO
CHANG, C H JOSEPH, KU MED CENTER,66103
588-6807
29 M 58301 53 R
CHIN, TOM D, KUMC - HUMAN ECOLOGY DEPT,66103
588-7175
22 M 2501 43 ID
CHO, CHENG T, K U MED CENTER,66103
588-6336
37 M 38501 62 PD
CHONKO, ARNOLD M, KU MED CENTER,66103
588-6969
43 M 3840 69 NEP
CHRYSANT, STEVEN G, KU MED CENTER,66103
861-4700
34 M 41801 59 CD
COALE, LLOYD H, 5020 GREELEY,66104
-
13 M 1902 43 OO
COCK, JAMES D, KU MED CENTER,66103
588-6031
36 M 6505 60 HEM
CODKE, ALLAN R, KUMC DEPT OF MED,66103
588-6990
36 M 14303 58 GE
COONFIELD, JAMES W, 1428 S 32ND,66106
384-1630
45 M 1902 78 FP
COX III, IRA L, 155 SOUTH 18TH,66102
371-4343
43 M 1902 68 DR
COX JR, WALLACE F, 4631 ORVILLE,66102
287-3600
30 M 1902 56 GS
CROCKETT, CHARLES A, 155 S 18TH,66102
342-2200
19 M 401 44 OPH
CROSS, BETTY S, 4031 SPRINGFIELD,66103
-
57 F 1902
CULP, LOUIS M, 1645 WASHINGTON BLVD,66102
371-1077
24 M 1902 53 FP
CURTIS, MIRJANA R, KU MED CENTER - OB/GYN,66103
588-6237
46 F 95702 69 OBG
CZAPANSKY, DESIREE K, 3838 RAINBOW APT 208,66103
-
59 F 1902
DARR, RICHARD B, 320 TERRACE TR WEST,66106
676-2097
42 M 3401 70 IM
DAVIS, CHRISTOPHER G, 219 HURON BLDG,66101
321-9313
09 M 1902 39 FP
DAY, HUGHES W, BETHANY MED CENTER,66102
281-8856
15 M 1902 39 IM

DE SMET, ARTHUR AUGUST, KU MED CENTER - DIAG RAD,66103
588-6800
47 M 2501 72 DR
DE VRIES, PIETER A, KU MED CTR PEDIATRIC SURG,66103
588-6180
21 M 511 47 PDS
DEITZ, MICHAEL R, 155 S 18TH,66102
342-2222
32 M 4101 58 OPH
DEMOTT, WAYNE R, PROVIDENCE-ST MGT HLTH CT,66112
334-2500
34 M 4002 59 PATH
DIALLO, GASTON I, 600 NEBRASKA,66117
281-2888
35 M 86905 64 GE
DICK, ARTHUR R, KU MEDICAL CENTER,66103
588-6985
34 M 2301 65 N
DIEDERICH, DENNIS A, K U MEDICAL CENTER,66103
588-6981
36 M 2834 61 IM
DUJOVNE, CARLOS A, K U MED CENTER,66103
588-6026
37 M 13201 61 CP
DULIN, JOSE I, 6013 LEAVENWORTH RD,66104
299-0089
51 M 84711 75 IM
DUNN, MARVIN I, KU MED CENTER,66103
588-6015
27 M 1902 54 CD
EGEA, FERNANDO M, 8919 PARALLEL PARKWAY,66112
334-3400
37 M 13206 62 N
EULBERG, JEANNE G, KU MED CENTER PEDIATRICS,66103
588-5908
49 F 3005 77 PD
FABIAN, CAROL J, K U MED CENTER,66103
588-6029
46 F 1902 ON
FITZPATRICK, M ROBERT, 1610 WASHINGTON BLVD,66102
321-4800
20 M 1803 44 FP
FLOERSCH, HUBERT M, 8919 PARALLEL PARKWAY,66112
371-2020
08 M 1902 35 OBG
FORET, JOHN D, KU MED CENTER,66103
588-6147
26 M 1602 53 U
FOX, DEANNA K, K U MED CENTER,66103
588-6670
48 F 1902 74 ANES
FOX, HOWARD A, K U MED CENTER,66103
588-6337
33 M 3501 62 PD
FRANCISCO, W DAVID, 155 S 18TH,66102
371-6802
21 M 1902 44 ORS
FRIESEN, STANLEY R, KU MED CENTER,66103
588-6108
18 M 1902 43 GS
GERJARUSAK, PRAPAS, 8919 PARALLEL SUITE 208,66112
788-9604
46 M 89101 71 IM
GILHOUSEN, FREDERIC M, 1029 N 32ND,66102
281-5252
40 M 1902 66 ORS
GIRON JR, LOUIS T, K U MED CENTER DEPT OF N,66103
588-6970
43 M 2802 68 N
GODFREY, WILLIAM A, KU MED CENTER,66103
588-6600
38 M 1902 65 OPH
GOERTZ, KENNETH K, 222 HC MILLER KUMC,66103
588-6311
50 M 1902 75 PD
GOERTZ, LEO R, 155 S 18TH ST,66102
371-4343
22 M 1902 52 R
GOODWIN, DONALD W, KU MED CENTER,66103
588-6402
31 M 1902 64 P
GOTO, HIROSHI, KUMC - ANES DEPT,66103
588-6670
42 M 57241 67 ANES
GRADY, KENNETH L, 155 S 18TH,66102
321-3844
36 M 1902 69 P
GRANTHAM, JARED J, K U MED CENTER,66103
588-6983
36 M 1902 62 NEP
GREENBERGER, N J, KU MEDICAL CENTER,66103
588-6001
33 M 3806 59 IM

GRUENDEL, RICHARD A, 1029 N 32ND,66102
281-5252
29 M 1902 55 DRS
GRUENDEL, VIRGINIA T, 6926 GARFIELD,66102
299-2787
30 F 1902 55 FP
GUARDIA, DAVID K, KU MED CENTER DEPT OB/GYN,66103
588-6243
52 M 1902 77 OBG
GUMUCIO, MARID L, 6013 LEAVENWORTH RD,66104
299-2069
30 M 64901 63 IM
HALL III, THOMAS BRYAN, KU MED CENTER,66103
588-6400
43 M 2802 69 P
HAMILL, JOHN M, 3828 BOOTH APT 2,66103
-
59 M 1902
HANCOCK, ALAN C, 9201 PARALLEL,66112
299-1474
35 M 1902 65 FP
HANSON, FREDERICK, K U MED CENTER,66103
588-6044
48 M 506 74 IM
HARA, GLENN S, KU MED CENTER,66103
588-6200
43 M 514 69 OBG
HARDIN, CREIGHTON A, KU MED CENTER,66103
588-6106
18 M 5605 43 GS
HARPER, DIANE M, 4136 BOOTH,66103
-
58 F 1902
HARRIS, JO ANN SPEIGEL, K U MED CENTER,66103
588-2773
50 F 3901 76 PD
HART, KELLY Z, 155 S 18TH,66102
371-4343
50 M 1902 75 OR
HARTMAN, CHARLES R, K U MED CENTER,66103
588-6111
37 M 1902 66 IM
HARTMAN, GERALD V, KUMC,66103
588-6815
20 M 1902 45 TR
HENNING JR, HAROLD JOHN, 4116 BOOTH,66103
-
55 M 1902
HERMRECK, ARLO S, K U MED CENTER,66103
588-7232
38 M 1902 65 GS
HETTINGER, MICHAEL E, KU MED CENTER,66103
588-6600
46 M 4706 75 OPH
HIEBERT, JOHN M, KU MED CTR PLASTIC SURG,66103
588-6143
42 M 2405 67 PS
HINTHORN, DANIEL R, KU MED CENTER,66103
588-6035
41 M 1902 67 IM
HOADLEY, WILLIAM D, KU MED CENTER-MEDICINE,66103
588-3974
31 M 1902 56 IM
HODGES, GLENN R, K U MED CENTER,66103
588-6035
41 M 1602 67 ID
HOLOCRAFT, JACQUELYNE, 4631 ORVILLE SUITE 203,66102
321-1161
36 F 2105 63 ENT
HOLMES, FREDERICK F, KUMC,66103
588-6987
32 M 5404 57 IM
HOLMES, GRACE E, KUMC,66103
588-6325
32 F 5404 57 PD
HOLMES, JOHN A, 155 S 18TH,66102
621-1188
47 M 1902 77 IM
HORTON, WILLIAM A, K U MEDICAL CENTER,66103
588-6043
45 M 1902 71 IM
HUERTER, QUENTIN C, WYANDOTTE MED BLDG STE226,66112
299-8800
31 M 1902 59 OPH
HURWITZ, ARYEH, KU MED CENTER,66103
588-6026
36 M 2802 61 IM
IBARRA, RICHARD C, 754 PACIFIC,66101
342-3969
26 M 64902 57 FP

ILIOPOULOS, JOHN I, KU MED CENTER,66103
588-6197
44 M 41801 69 GS
INGRAM, JOHN E, 1428 S 32ND,66106
384-1630
24 M 3006 56 FP
JACOBS, DAVID S, 8929 PARALLEL PARKWAY,66112
334-2500
31 M 2501 56 PATH
JACOBS, RAE R, K U MED CENTER,66103
588-6163
36 M 3506 62 OR
JAHANIAN, DARYOUSH, 8919 PARALLEL PARKWAY,66112
371-2020
40 M 51701 64 OBG
JAYARAM, MARANDAPALLI R, 8919 PARALLEL,66112
492-6200
42 M 49509 65 PD
JEWELL, WILLIAM R, KU MED CENTER,66103
588-6112
35 M 1611 61 GS
JOHNSON, BRUCE E, KU MED CENTER,66103
588-6000
50 M 514 76 IM
JOHNSON, CYNOA A, KU MED CTR FAMILY PRACTIC,66103
588-1908
51 F 514 77 FM
JOHNSON, JOHN E, BETHANY MED CTR,66102
281-8815
17 M 4706 43 PATH
JONES JR, HERMAN H, 600 NEBRASKA,66101
342-4010
25 M 4707 54 GS
KALIVAS, JAMES T, KU MED CENTER,66103
588-6028
38 M 502 63 O
KANNEL, PHILIP O, KU MED CENTER OTORHINOLAR,66103
588-6700
50 M OTO
KARNAZE, MICHAEL G, 1900 WASHINGTON AVE,66102
-
56 M 1902
KEELER, LINDA L, KU MED CTR DEPT OF PSY,66103
588-6475
47 F 1902 76 P
KENNEDY, JAMES A, K U MED CENTER,66103
588-6000
35 M 2834 61 IM
KEPES, JOHN J, K U MED CENTER - PATH DEP,66103
588-7076
28 M 47301 52 PATH
KERBY, GERALD R, KU MED CENTER,66103
588-6044
32 M 1902 58 PUD
KESTENBAUM, THELDA M, K U MED CENTER,66103
588-6028
48 F 5101 73 O
KIM, JONG M, KUMC,66103
588-6670
40 M 58302 64 ANES
KING, CHARLES R, K U MEDICAL CENTER,66103
588-6200
47 M 1902 71 OBG
KING, TERESA M, 4309 ADAMS,66103
384-5237
59 F 1902
KIRCHNER, FERNANDO R, 155 S 18TH SUITE 270,66102
371-7333
30 M 64901 55 OTO
KOVAC, ANTHONY L, KU MED CENTER ANES DEPT,66103
588-6670
52 M 1902 77 ANES
KRANTZ, KERMIT E, KU MED CENTER,66103
588-6201
23 M 1606 48 OBG
KUEBLER, KEVIN M, 155 S 18TH,66102
371-6800
50 M 2101 75 GS
KWEE, SIDE T, PROVIDENCE-ST MGT HLTH CT,66112
334-2500
36 F 1720 63 PATH
KYNER, JOSEPH L, KU MED CENTER,66103
588-5554
34 M 1902 60 END
LAI, CHI-WAN, K U MED CENTER DEPT OF N,66103
588-6014
44 M 38502 69 N
LAING, ROBERT R, 155 S 18TH ST,66102
371-4301
37 M 1643 61 GE
LAWHORN, STEPHANIE LU, 3909 BOOTH APT 9,66103
722-0691
54 F 1902

LAWWILL, THEODORE, K U MED CENTER,66103
588-6605
37 M 4705 61 OPH
LAYBOURNE JR, PAUL C, KU MED CENTER,66103
588-6475
19 M 3509 44 CHP
LEE, JAE M, 155 S 18TH #105,66102
677-3555
40 M 58302 65 GS
LEE, KYO R, KUMC,66103
588-6800
33 M 58302 59 R
LEE JR, JAMES G, 5739 METCALF CT,66202
371-2330
18 M 1902 44 OBG
LEMOINE JR, ALBERT N, K U MED CENTER,66103
588-6600
18 M 2802 43 OPH
LEO, WILLIAM A, K U MED CENTER,66103
588-6109
22 M 1902 48 GS
LEVINE, ERROL, K U MED CENTER,66103
588-6800
41 M 83601 64 DR
LIEBERMAN, BRUCE IRWIN, KU MED CENTER,66103
588-6300
49 M 3819 74 PD
LINDSLEY, CAROL B, K U MED CENTER,66103
588-5907
41 F 5404 68 PD
LINDSLEY, HERBERT B, K U MED CENTER,66103
588-6008
40 M 1902 66 RHU
LINN, CATHERINE P, 155 S 18TH,66102
788-9797
52 F 1902 77 OBG
LINSHAW, MICHAEL A, K U MED CENTER PED DEPT,66103
588-6323
40 M 4109 66 PD
LIU, ALBERT T, 8919 PARALLEL, 66112
788-9797
49 M 1902 79 OBG
LIU, CHIEN, K U MED CENTER,66103
588-6035
21 M 24217 47 IO
LLOYD, HARVEY L, PO BOX 6037,66106
-
08 M 1803 36 FP
LOTITO, CARLOS A, 4631 ORVILLE,66102
596-1185
29 M 13201 56 IM
LOWMAN, JAMES T, KUMC,66103
588-5701
31 M 401 58 PD
LUKERT, BARBARA P, KU MED CENTER,66103
588-6048
34 F 1902 60 END
LYNCH, SEAN R, KU MED CENTER,66103
588-6031
38 M 83601 61 HEM
MACARTHUR, RICHARD IAN, KUMC 39TH AT RAINBOW,66103
588-6100
46 M 1902 73 GS
MAGRINA, JAVIER F, K U MED CENTER OBG DEPT,66103
588-6244
49 M 84701 68 OBG
MANGOLD, JOEL JOYCE, KU MED CENTER,66103
588-6670
50 M 1902 76 ANES
MANI, MANI M, KUMC,66103
588-6136
37 M 49527 60 PS
MANSFIELD, CARL M, K U MED CENTER - RM 117B,66103
588-7350
28 M 1003 56 TR
MARPLES, BRADLEY W, 3904 ADAMS,66103
-
56 M 1902
MARSH, ALICE GARRISON, K U MED CENTER,66103
588-6390
24 F 3545 49 PD
MARTIN, JOSEPH P, 9201 PARALLEL,66112
334-1515
49 M 1902 74 IM
MARTIN, NORMAN L, K U MED CENTER,66103
588-6800
36 M 1902 62 OR
MARTIN, RONALD L, KU MED CENTER PSYCHIATRY,66103
588-6412
45 M 1606 71 P
MASTERS, FRANCIS W, KU MED CENTER,66103
588-6142
20 M 3545 45 PS

MATHEWSON, HUGH S, KUMC,66103
588-6675
21 M 1902 44 ANES
MATTIOLI, LEONE, KUMC,66103
588-6311
32 M 56115 56 POC
MC PHEE, MARK S, KU MED CENTER-MEDICINE,66103
588-6001
51 M 1902 76 IM
MCCARTHY, ROBERT P, 155 S 18TH,66102
342-7233
25 M 2834 53 U
MCCUNE, MARK A, 8919 PARALLEL PKWY,66112
492-6200
52 M 1902 77 O
MEBUST, WINSTON K, KU MED CENTER,66103
588-6148
33 M 5404 58 U
MEDHAT, MOHAMED A, KU MED CENTER REHAB MED,66103
588-6787
32 M 33002 54 RM
MEEK JR, JOSEPH C, KU MED CENTER,66103
588-6023
31 M 1902 57 IM
MESINA, ROLANDO R, 1200 N 38TH,66102
371-3829
37 M 74801 61 GS
MILLIGAN, DONALD B, KU MED CENTER,66103
588-1937
48 M 2307 74 FP
MINER JR, PHILIP B, K U MED CENTER,66103
588-6990
46 M 702 71 GE
MOELLER, DONALD D, 155 S 18TH,66102
371-4301
34 M 1902 60 GE
MOORE, WAYNE V, K U MED CENTER,66103
588-6336
42 M 2604 70 PD
MORFFI, RAUL R, 1225 N 78TH,66112
287-8300
25 M 27501 51 IM
MULLEN SR, CLIFFORD J, 1828 WASH BLVD,66102
-
98 M 3006 23 OD
NEFF, JAMES R, KU MED CENTER,66103
588-6198
40 M 1902 66 ORS
NEIGHBOR, ERNEST G, 1420 S 42ND,66106
-
06 M 1902 33 FP
NEIGHBOR, GAYLORD P, 1420 SOUTH 42ND,66106
831-1100
13 M 1902 41 FP
NELSON, WILLIAM PAUL, KU MED CENTER,66103
588-6015
30 M 1002 56 CO
NEWKIRK, DARREL D, 2427 N 73RD,66109
321-4803
42 M 2101 67 PH
NIBBELINK, LARRY WAYNE, 155 S 18TH,66102
371-2330
48 M 2803 75 OBG
NOBLE, MARK J, KU MED CENTER-UROLOGY,66103
588-6148
49 M 2501 75 U
NOHE, PHILIP C, 266 NEW BROTHERHOOD BLDG,66101
-
17 M 1902 42 OD
NORRIS, CHARLEY W, KUMC,66103
588-6700
33 M 1902 64 OTO
NOTHNAGEL, ARNOLD F, 501 WESTVALE,66102
-
15 M 1902 39 OD
NUNEZ, JULIAN, 4631 ORVILLE #209,66102
299-2088
30 M 27501 60 FP
O'BOYNICK II, PAUL LEONARD, KU MED CENTER,66103
588-5000
48 M 1902 73 NS
OLDFIELD, RAY W, 4631 ORVILLE,66102
287-3537
45 M 1902 72 FP
OTHMER, EKKEHARD, K U MEDICAL CENTER,66103
588-6440
33 M 40721 66 P
OVERFIELD, A SCOTT, 3838 RAINBOW #906,66103
-
57 M 1902
OXLER JR, JOHN EDWARD, 155 S 18TH,66103
362-6161
46 M 1902 72 IM

PALMER, MARVIN M, 8919 PARALLEL PKW,66112
788-9797
45 M 702 71 OBG
PALMER1, MAR1A, 4631 ORVILLE,66102
596-1185
49 F 13201 75 FP
PALMER1, V CARLDS, 4631 DRV1LLE,66102
596-1185
46 M 13201 75 FP
PAROD, MANUEL P, K U MED1CAL CENTER,66103
588-6464
35 M 74801 62 P
PAREKH, AJ1TKUMAR M, 6013 LEAVENWORTH RD,6610C4
299-2069
47 M 49501 71 PUO
PARK, CHAN H, K U MEDICAL CENTER,66103
588-6029
36 M 58302 62 1M
PARRA, M1GUEL O, 6013 LEAVENWORTH RD,66104
299-2088
37 M 84710 64 FP
PASCH1NO, ANTCN1D R, 4631 ORVILLE #215,66102
287-7100
22 M 13201 60 P
PAZELL, JOHN A, 4631 ORVILLE,66102
287-6464
40 M 2501 66 DRS
PECANA, MANUEL C, BETHANY MED CENTER,66102
281-8896
45 M 74801 69 ANES
PENNER, T1MOTHY M, 3700 CAMBR1OGE,66103
-
59 M 1902
PERRY JR, LAWRENCE L, KUMC 39TH AT RA1NBOW,66103
588-6522
34 M 1902 59 FP
P1ERCE, GEORGE E, KUMC - PD BOX 255,66103
588-6115
33 M 2307 60 TS
P1NGLETON, SUSAN K, KU MED CENTER DEPT OF MED,66103
588-6044
46 F 1902 72 1M
P1PP1N, LYNNE K, KU MED CENTER ANES,66103
588-6670
48 F 35207 72 ANES
P1SCHKE, FRANK J, 155 S 18TH,66102
321-1161
36 M 1902 62 OTO
POL, P ALBERT, 6013 LEAVENWORTH RD,66104
287-7100
36 M 84710 67 P
PORTER, DAV1D M, 4517 TRDUP,66102
287-8800
39 M 4707 64 PD
PDTTER, RDBERT L, 1969 N 33RD,6610C2
321-0341
38 M 1902 64 1M
POWERS, G RDBERT, 8919 PARALLEL PKWY,66112
299-1469
33 M 1902 65 FP
PREMS1NGH, NAL1NI G, 4631 ORVILLE #202,66102
596-2000
39 F 49508 65 CD
PRESKORN, SHELDON H, K U MED CENTER,66103
588-6400
48 M 1902 74 P
PRESTON, DAV1D F, KU MED CENTER,6610C3
588-6810
33 M 3841 59 NM
PRETZ, JAMES B, 1610 WASH1NGTDN BDULEVARD,66102
342-2442
24 M 1902 47 FP
PR1CE, H1LTON 1, KU MED CENTER - RAD DEPT,66103
588-6831
49 M 83601 72 DR
PRICE, JAMES GORDON, K U MED CENTER,66103
588-6510
26 M 702 47 FP
PR1ETO, JDRGE N, 6013 LEAVENWORTH RD,66104
299-2069
45 M 26401 69 GS
PRDCHASKA, MARK L, RA1NBDW TOWERS APT 1206,66103
-
57 M 1902
PROUD, G DNE1L, KU MED1CAL CENTER,66103
588-6700
13 M 2802 39 DTO
PUGH, DAV1D M, K U MED CENTER,6610C3
588-6015
29 M 801 58 CO
OU1NN, CHARLES E, 4601 ORVILLE,66102
287-6604
43 M 4707 68 OBG

RAGLAND, CHARLES W, 155 S 18TH,66102
321-2974
50 M 1902 77 1M
RECKL1NG, FREDER1CK W, KUMC,66103
588-6129
34 M 3545 59 ORS
REDDY, EASHWER K, K U MED CENTER - RAD DEPT,66103
588-7350
44 M 49597 68 TR
REDFORD, JOHN W B, K U MED CENTER,66103
588-6777
28 M 6501 53 PM
REEB, RONALD JOSEPH, 155 S 18TH,66102
371-4343
46 M 3006 72 OR
REED, JAMES STEWART, K U MED CENTER,66103
588-6019
46 M 3601 72 GE
REILLY, DOUGLAS K, KU MED CTR PSY DEPT,66103
-
46 M 3005 74 P
RHODES, JAMES B, KU MED1CAL CENTER,66103
588-6019
28 M 1902 58 GE
RHODES, MARTIN L, 155 S E1GHTEENTH,66102
321-0341
47 M 1902 72 1M
R1CE JR, FREDER1CK A, 1029 N 32ND ST,66102
281-5252
36 M 4802 63 DRS
RICHARDSON, JAY L, 8919 PARALLEL, 66112
299-8000
38 M 1902 65 GS
R1LEY, RAY B, 2020 ORV1LLE,66102
-
06 M 1902 36 DO
R1NKENBERGER, ROBERT, K U MED CENTER,66103
588-6015
44 M 1902 71 CO
R1SING, JESSE O, KU MED1CAL CENTER,66103
588-1934
14 M 1902 38 1M
ROB1NSON, DAV1D W, KU MED1CAL CENTER,66103
588-6136
14 M 4101 38 PS
RDB1NSDN, RALPH G, KU MED1CAL CENTER,66103
588-6810
37 M 1902 62 NM
ROORIGUEZ, RAUL G, 6013 LEAVENWORTH RD,66104
299-2525
42 M 26401 68 P
ROOK, LEE E, 4116 STRONG,66106
831-2834
09 M 1902 38 FP
ROSENTHAL, STANTON J, K U MED CENTER,66103
588-6800
46 M 1902 71 OR
RDSS, STEVEN E, KU MED CENTER SURG DEPT,66103
588-6150
51 M 4102 76 GS
ROTH, ALAN E, BETHANY HOSP1TAL 51 N 12,66102
281-8814
35 M 1902 62 PATH
RUB1N JR, BEN, 132 S SEVENTEENTH,6610C2
371-2561
37 M 3005 61 PD
RUSSO, LIBBIE V, KU MED CENTER, 66103
588-6326
53 F 2846 77 PDE
RUTH, WILL1AM E, K U MED CENTER,66103
588-6044
26 M 1902 53 PUD
RYAN, M1CHAEL J, 764 NEW BROTHERHOOD BLDG,66101
342-7070
14 M 2834 37 OTD
SAVIN, VIRG1N1A J, KU MED CENTER,66103
588-6983
44 F 4112 70 IM
SCH1MKE, R NE1L, KU MED CENTER,66103
588-6043
35 M 1902 62 IM
SCHDTLAND, EDWARD S, 1300 N 78TH STE 303,66112
334-5250
36 M 86901 66 O
SCHREPFER, RDSEMARY, KU MED CENTER,66103
588-6200
22 F 1902 47 DBG
SCHWEGLER, RAYMOND A, 8919 PARALLEL PKWY,66112
492-6200
37 M 1902 63 CD
SCHWORM, CURTIS P, 155 SOUTH 18TH,66102
371-4343
47 M 3005 73 DR

SERERES, EDGAR P, 733 N 75TH TERR,66112
299-9010
15 M 1902 39 FP
SHERMAN, ROBERT P, 9201 PARALLEL AVE,66112
334-0040
34 M 1902 63 PUD
SIFERS, EARL C, 155 S 18TH,66102
371-2900
24 M 1902 47 GS
SIFERS, TIMOTHY M, 155 S 18TH,66102
371-2900
48 M 1902 74 GS
SILVERS, ALVIN, 1702 SW BOULEVARD,66103
236-9691
18 M 1902 45 FP
SKIKNE, BARRY S, KU MED CENTER,66103
588-6031
45 M 83601 61 HEM
SMITH, WILLIAM P, 7312 ANTIOCH,66204
677-0883
51 M 1902 77 R
SNODGRASS, WAYNE R, KU MED CENTER PED DEPT,66103
588-6318
45 M 1720 68 PD
SOUCEK, CHARLES D, 155 S 18TH,66102
371-4343
31 M 3005 56 R
SOUTHWICK, CHRISTOPHER L, PO BOX 3436,66103
-
56 M 1902
SPEER, LELAND, 910 N WASHINGTON,66102
-
12 M 1902 36 OD
STECHSCHULTE, DANIEL J, K U MED CENTER,66103
588-6008
36 M 2834 62 A
STEELE, CLARENCE H, 255 BROTHERHOOD BLDG,66101
321-1161
14 M 1902 40 DTD
STEINZEIG, SHERMAN M, 155 S 18TH,66102
621-1151
25 M 1902 52 CD
STEPHENS, RONALD L, KUMC,66103
588-6029
39 M 1902 65 IM
STUBBLEFIELD, CHARLES T, 155 S 18TH,66102
371-2330
32 M 1902 58 OBG
TAYLOR, SARAH A, KU MED CENTER-STUDENT UN,66103
588-6029
50 F 1902 75 IM
TAYLOR, THOMAS L, 155 S 18TH ST,66102
371-2900
40 M 1902 66 GS
TAYLOR, WILLIAM F, 8919 PARALLEL PKWY,66112
299-8000
53 M 2846 76 IM
TEMPLETON, ARCH W, K U MEDICAL CENTER,66103
588-6805
32 M 3005 57 R
THERON, LEONA F, K U MED CENTER PED DEPT,66103
588-5908
41 F 6701 67 PD
THOMAS, JAMES H, K U MED CENTER,66103
588-5901
41 M 2012 66 GS
THOMAS, THOMAS V, 211 INDIAN SPRINGS MED BL,66102
287-2600
37 M 49549 61 CDS
THOMPSON, DANNIE M, ONE GATEWAY CENTER,66101
321-3355
35 M 4707 64 OBG
TOSONE, STEVEN R, K U MED CENTER,66103
588-6670
52 M 1902 77 ANES
TRUEWORTHY, ROBERT C, KU MED CENTER,66103
588-6340
40 M 2802 66 PD
UTLEY, JAMES HARMON, 51 N 12TH,66102
281-8880
51 M 1606 74 EM
VAN THULLENAR, PHILIP A, BETHANY MED CENTER,66102
281-8834
31 M 2834 57 PATH
VARGHESE, GEORGE, K U MEDICAL CENTER,66103
488-6798
44 M 49509 69 PM
VATS, TRIBHAWAN S, K U MED CENTER PED DEPT,66103
588-6340
40 M 49529 63 PD
VELARDE, HUGO, 4601 ORVILLE AVE STE 14,66102
287-8400
M 64 GS

WAODELL, BILL O, 155 SOUTH 18TH,66102
321-0386
31 M 3901 56 IM
WALKER, JACK O, KU MED CENTER,66103
588-1900
22 M 1902 53 FP
WALKER, MAURICE A, 3214 STRONG AVE,66106
831-1433
04 M 1601 28 GS
WALSH, DAVID J, KU MED CENTER PED DEPT,66103
588-6371
46 M 4501 73 PD
WALTER, DONNAL C, KU MED CENTER,66103
588-6337
48 M 1902 76 PD
WALTERS, WILLIAM DAVID, 1428 S 32ND,66106
384-1630
50 M 1902 78 FP
WAXMAN, DAVID, K U MED CENTER,66103
588-1207
18 M 3515 50 IM
WEIGEL, JOHN W, UROLOGY DEPT KU MED CTR,66103
588-6148
29 M 1902 54 U
WEINSTEIN, GARY L, 3824 BOOTH #9,66103
677-1563
M 1902
WELLER, ELIZABETH B, KU MED CENTER,66103
588-6464
49 F 60501 75 CHP
WELLER, RONALD ALAN, KU MED CENTER - PSY DEPT,66103
588-6464
48 M 2802 74 P
WILEY, THOMAS M, 4150 BOOTH,66103
-
59 M 1902
WILLIAMS JR, STERLING B, K U MEDICAL CENTER,66103
588-6200
41 M 401 73 DBG
WISE III, JOSEPH EDWARD, 132 S 17TH,66102
371-2561
51 M 1902 76 PD
WISNER JR, JOHN HENRY, 229 S EIGHTH,66101
588-6431
46 M 1902 76 P
WOLF, KARL T, 621 NORTHRUP AVE,66101
-
14 M 1902 48 OD
WONDER II, DONALD K, 2904 W 46TH,66103
-
53 M 4802 79 GS
WONG, BERT Y, K U MED CENTER,66103
588-6015
40 M 801 65 CD
WOOD, FRED M, KU MED CENTER,66103
588-6144
38 M 4706 62 ORS
WRIGHT III, ROBERT W, 4606 CAMBRIDGE,66103
588-6670
50 M 1902 79 ANES
WRIGHT JR, ROBERT W, 669 NEW BROTHERHOOD BLDG,66101
371-5344
24 M 1902 48 GS
YAKAR, DANIEL, KU MED CTR RAD DEPT,66103
588-3600
32 M 55001 58 RT
ZAREMSKI, SHERMAN C, 4631 ORVILLE STE 209,66102
596-1185
33 M 1720 58 IM
ZIEGLER, DEWEY K, KU MED CENTER,66103
588-6985
20 M 2401 45 N
ZINN, THOMAS W, 155 S 18TH,66102
371-4343
41 M 1902 67 R

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ALLEN, MARK LYNN, 3720 WYOMING SUITE 3,64111
-
53 M 1902 80 ANES
ANDERSON, WILLIAM A, 629 WEST 70TH,64113
234-3079
50 M 2846 76 EM
BENNETT, CHARLES A, 5619 NW HILLSIDE DR,64151
-
96 M 1902 25 DO
BOUDET, ROBERT A, V A HOSP,64128
861-4700
36 M 1103 62 GS

CALKINS, W GRAHAM, V A HOSP,64128
861-4700
26 M 2501 50 GE
CHRISTENSEN, SHANE R, 4812 HEINTZ, 64133
281-8881
55 M 1902 79 EM
CRABB, JEREMIAH E, 5924 GRAND, 64113
588-6044
54 M 1902 79 IM
DAVIS JR, JAMES W, V A HOSP,64128
861-4700
33 M 4705 57 IM
DIEHL, ANTONI M, 4320 WORNALL RD,64111
753-4414
24 M 2604 47 POC
FALLON, JOHN H, 3730 WYOMING,64111
-
59 M 1902
FESTOFF, BARRY W, VA HOSPITAL,64128
861-4700
40 M 1102 77 N
GLATTER, THOMAS R, VA HOSPITAL,64128
861-4700
34 M 1611 60 IM
GODFREY, ROBERT G, VA HOSP,64128
861-4700
27 M 1902 58 RHU
HARO, BENJAMIN F, 8400 HAWTHORN RD,64120
242-2674
28 M 4802 55 OBG
HOHLY, EVE K, 3923 HARRISON,64110
-
40 F 1902
HYMAN, HENRY T, O.D., 5140 NE ANTIOCH RD,64119
-
49 M 2879 76 OBG
JETER, JOHN HORTON, 2301 HOLMES,64108
556-3000
55 M 1902 81 EM
JOHNS JR, LEO E, VETERANS MED CENTER,64128
861-4700
22 M 2101 45 PUD
JOHNSON, FREDERICK E, C/O MARY K BLOTT,64112
-
92 M 2843 21 DD
KINPORTS JR, EDWARD B, 2400 PERSHING RD STE 561,64108
842-1950
46 M 3006 74 EM
KINPORTS SR, EDWARD B, 2520 GRAND AVE #309,64108
-
15 M 1602 42 EM
KRUEGER, KURT ALLEN, 10002 HOWE DR,66206
648-0323
48 M 3006 74 ANES
LEWIS JR, H DANIEL, VETERANS MEDICAL CENTER,64128
861-4700
32 M 5101 58 CO
LUETJE, CHARLES MARION, 2928 MAIN SUITE 105,64108
561-4423
41 M 2803 67 DTD
MCVEY, PAMELA S, #1 CROWN CENTER,64108
842-1950
56 F 1902 80 EM
ORR, STEVEN M, 4615 NE WINN RD #202,64117
-
55 M 1902 79 EM
PAYNE, J RALPH, 4460 ROCKHILL TERRACE,64110
334-2500
40 M 1902 62 EM
PERICO, CARLOS J, 5801 E 113TH,64134
761-1866
32 M 26404 60 FP
REDING, DOUGLAS J, 428 W 60TH TERR,64113
588-5000
54 M 1803 80 IM
SALEH, GEORGE A, O.D., 5140 NE ANTIOCH RD,64119
452-7300
50 M 2878 OBG
SINGER, PHILIP A, V A HOSPITAL,64128
861-4700
42 M 3545 69 N
SLEEPER, CAROL A, 20 E FOURTEENTH,64142
-
33 F 1902 58 PATH
SMITH-HOLMES, BARBARA K, 1147 EAST 77TH,64131
556-3100
55 F 1902 80 EM
STASS-ISERN, MERRILL, 1405 W 50TH TERR,64112
281-8880
50 M 84706 77 EM
STOKES, ROBERT LEE, 3621 BELLEVIEW,64111
281-8882
43 M 4804 74 EM
VITALE, NEIL B, 1135 W 41ST,64111
-
55 M 1902
WESSELIUS, LEWIS J, VA HOSPITAL,64128
861-4700
50 M 1902 82 PUD
WIEGMANN, THOMAS B, VETERANS MED CENTER,64128
-
43 M 40902 69 NEP
WIENS, TIMOTHY K, 3539 GENESSE,64111
-
55 M 1902

KINGMAN—316
(Ninnescah Society)

BLOOM, L THEIL, BOX 496,67068
-
32 M 1902 57 R
BOYER, ROBERT E, 760 AVENUE O WEST,67068
532-5145
36 M 1902 63 FP
BURKET JR, GEORGE E, SPRING LAKE RTE 1,67068
-
12 M 1902 37 DD

KINSLEY—316
(Edwards County)

ATWOOD, M DALE, PO BOX 269,67547
659-2114
19 M 1902 51 FP
MCKIM, W LYNN, 109 WEST 8TH,67547
659-2137
33 M 1902 59 FP
SCHNOEBELEN, RENE E, 807 EAST 4TH,67547
659-2141
16 M 3901 40 FP

KIOWA—316
(Tri-County Society)

CHRISTENSEN, MARION O, 220 SOUTH EIGHTH,67070
825-4121
25 M 3901 52 FP
RODRIGUEZ, ALBERTO, 425 CAMPBELL,67070
825-4039
25 M 27501 49 GP

LA CROSSE—913
(Central Kansas Society)

BHARGAVA, ASHOK KUMAR, BHARGAVA CLINIC,67548
222-2564
37 M 49547 64 FP
BHARGAVA, SHOBHANA, BHARGAVA CLINIC,67548
222-2564
38 F 49547 64 FP

LANSING—913
(Leavenworth County Society)

SNOW, DONALD L, HOLIDAY PLAZA,66043
727-3204
21 M 64901 54 OBG

LARNED—316
(Pawnee County Society)

BRENNER, WILLIAM R, 1031 W FOURTH,67550
285-3133
15 M 3006 39 FP
CRAM JR, OLE R, 722 MANN,67550
285-2141
18 M 1902 43 FP
DAVIS, DAVID H, 815 W 6TH,67550
-
04 M 1902 30 OO
EWING, THOMAS D, 804 CARROLL,67550
285-3133
22 M 1902 46 FP

PARAMESH, JAYA, ROUTE 3 BOX 89 LSH,67550
285-6285
32 F 49608 55 GP
SHAH, MIAN, SHAH CLINIC,67550
285-3173
32 M 70403 58 GS
SHAH, NASREEN, SHAH CLINIC,67550
285-3173
39 F 70409 62 OBG
SHEPARD, LEROY W, 603 W 5TH,6755C
-
04 M 3006 30 OO
SMITH, JOHN O, 804 CARROLL,67550
285-3133
22 M 3901 5I FP
WAGNER, LENARO D, 923 CARROLL,67550
-
54 M 512 79 FP

LAWRENCE—913
(Douglas County Society)

ANDERSON, WINSTAN L, 3603 W 10TH ST,66044
-
09 M 1902 34 OO
BAILEY, WILLIAM A, PO BOX 127,66044
843-9125
40 M 1902 66 ORS
BELOT JR, MONTI L, LAWRENCE NATIONAL BK BLDG,66044
843-3640
13 M 1902 40 FP
BIERI, PETER V, 1112 W SIXTH ST,66044
841-5217
45 M 1902 71 ENT
BISHOP, RODNEY LEE, LAWRENCE IM ASSOC,66044
842-7200
49 M 1902 75 IM
BITTENBENDER, LEE R, 930 IOWA,66044
842-7001
46 M 1902 72 O
BLAIR, T RICHARD, 404 MAINE STE 3,66044
842-3635
34 M 1902 60 IM
BOYDEN, MARY S, MEDICAL ARTS BLDG,66044
842-3778
14 F 2604 38 PDA
BRANSON, VERNON L, 346 MAINE,66044
842-4477
17 M 1902 42 PO
BRUNFELOT, JOAN KRAUS, 404 MAIN,66044
842-3635
52 F 1902 77 IM
BUCK JR, HENRY W, 1112 W SIXTH,66044
841-9200
34 M 1902 60 OBG
CARNAHAN, ROBERT L, 1112 W SIXTH SUITE 112,66044
841-4310
42 M 1902 70 IM
CHEDIAK, ELIAS, 601 MISSOURI,66044
841-7430
39 M 84704 65 P
CLINTON, DALE L, 15 E SEVENTH SUITE 103,66044
841-5716
21 M 1902 54 GP
CULVER, WARREN T, 2500 W 6TH,66044
842-4178
20 M 3508 46 OPH
DOUGLAS, JOSEPH MAHLO, 930 IOWA,66044
841-3535
39 M 1902 65 P
DUNLAP, RICHARD L, MEDICAL ARTS CENTER,66044
842-4344
12 M 3005 37 EENT
FRIESEN, DALE, PO BOX 521,66044
842-7026
47 M 1902 74 ANES
GILLES, HELEN M, MED ARTS CENTER,66044
842-4477
22 F 1902 45 PD
GOOWIN, PHILLIP A, 500 ROCKLEDGE,66044
841-6540
28 M 1902 55 ANES
GRAY, C K, LAWRENCE IM ASSOC,66044
842-7200
48 M 1902 75 IM
HAGGAN, MARGARET E, 1746 N H,66044
843-4455
F 2501 42 FP
HASSELLE III, JAMES E, 601 MISSOURI,66044
841-7430
35 M 4706 59 P

HATTON, DONALD W, 404 MAINE ST,66044
842-3635
42 M 1902 68 IM
HERMES, RICHARD L, THE MEDICAL ARTS CENTER,66044
843-0677
15 M 4112 39 OBG
HIEBERT, DAVID L, 1112 W SIXTH,66044
841-3211
36 M 1902 61 R
HIRO, WAYNE E, 2802 TRAIL RD,66044
842-4300
26 M 1902 50 TS
HUGHES, ROBERT WALTER, MEDICAL ARTS BLDG,66044
843-1374
27 M 1902 54 FP
INGHAM JR, H LAIRD, 404 MAINE,66044
842-3635
45 M 3901 70 IM
JONES, H PENFIELD, MED ARTS CENTER,66044
-
06 M 2401 31 GS
JOSEPH, HOWARD F, DOCTORS BUILDING,66044
843-3981
26 M 1902 51 U
LEARNED, GEORGE R, 401 ARKANSAS,66044
843-5502
22 M 1902 55 GS
LESSENDEN, GLENN A, 1112 W SIXTH SUITE 110,66044
843-6233
24 M 1902 48 FP
LOVELAND, G CHARLES, 346 MAINE,66044
842-4477
47 M 1902 73 PD
MADSEN, GLENN L, 1112 W SIXTH,66044
841-3211
38 M 3005 65 R
MANAHAN, G EUGENE, MED ARTS CENTER,66044
842-0211
19 M 1902 44 GS
MITCHELL, ALEX C, 1626 W 20TH,66044
843-4739
18 M 1902 50 PH
MODDRELL, CAROL A, 404 MAINE,66044
749-6100
45 F 1902 71 PATH
MONCKTON, LAURANCE A, 1112 W SIXTH SUITE 204,66044
843-2010
48 M 1902 74 GS
MYRICK, STEPHEN W, 346 MAINE,66044
842-6644
52 M 1902 77 GS
NELSON, RICHARD O, 935 IOWA,66044
843-0921
11 M 1001 41 FP
OELSCHLAGER, RONALD D, 1112 W SIXTH,66044
841-3211
43 M 1902 69 R
OLSON, CARL E, 935 IOWA,66044
842-9911
17 M 1611 46 FP
ORCHARD, RICHARD A, 1112 W SIXTH SUITE 212,66044
841-2280
41 M 2802 68 OPH
OSBERN, LIDA, 404 MAINE,66044
842-3635
52 F 1902 77 IM
OWEN, GARRY D, 1112 W SIXTH,66044
841-9200
37 M 1902 63 OBG
PEES JR, GERALD BOYD, 1112 W SIXTH SUITE 210,66044
843-5160
45 M 1902 71 IM
PLACEK, DEBRA C, MED ARTS CENTER,66044
843-0677
53 F 3005 78 OBG
PRAEGER, MARK A, 1112 W SIXTH SUITE 204,66044
843-2010
42 M 1902 68 GS
REED, JAMES S, WATKINS MEMORIAL HOSP,66045
-
23 M 1902 47 FP
REED, RALPH R, 404 MAINE,66044
842-3635
27 M 1902 53 IM
REESE, JOHN L, 346 MAINE,66044
842-6644
35 M 1902 61 GS
REITH, PAUL, 1331 NAISMITH,66044
-
M 2834 IM
ROBERTS, RICHARD S, 308 MAINE,66044
843-6137
19 M 2802 44 GS

SANDERS, J ALAN, LAWRENCE MEMDRIAL HDSP,66044
842-2083
29 M 1902 60 PATH
SCHNDSE, GREGORY D, 1112 W SIXTH SUITE 210,66044
843-5160
51 M 1902 76 IM
SCHRDEDER, SYDNEY O, WATKINS MEMDRIAL HDSP,66044
864-4045
18 M 1902 44 P
SCHWEGLER, RAYMDND A, 1504 UNIVERSITY DR,66044
-
07 M 2604 31 DD
SDSINSKI, RICHARD F, 1112 W SIXTH,66044
843-5160
51 M 1902 76 IM
STIVERS, MARK T, 404 MAINE,66044
842-2083
47 M 2803 73 PATH
TUCKER, DDNALD R, 1517 INDIAN WELLS CT,66044
354-5100
31 M 1902 57 IM
VERNDN, MARY C, 500 RDCKLEDGE,66044
841-6540
52 F 1902 77 FM
WELL, MICHAEL A, 944 KENTUCKY,66044
843-3981
41 M 1606 67 U
WERTZBERGER, JDHN, PD BOX 127,66044
843-9125
36 M 1902 63 DRS
WERTZBERGER, KENNETH LYNN, 1112 W SIXTH BDX 127,66044
843-9125
47 M 1902 73 DRS
WILCDX SR, HDWARD L, MEDICAL ARTS CENTER,66044
843-0677
18 M 3520 44 DBG
WDLLMANN, MARTIN, 2615 DRCHARD LN,66044
843-4455
26 M 1902 57 IM

LEAVENWORTH—913
(Leavenworth County Society)

AL-BAGHAL, MDHAMMAD, 113 DELAWARE SUITE A,66048
651-2200
45 M 87501 72 U
ALLEN JR, WILLIAM R, RT 3 BDX 295C,66048
-
46 M 1902 78 R
ANWAR, M ZIA, 109 DELAWARE,66048
651-2977
39 M 11801 67 IM
ASHKAR, ADNAN A, 109 DELAWARE SUITE F,66048
682-6818
42 M 52801 73 DBG
BARRY, DAVID R, 500 EISENHDWER RD,66048
727-6000
42 M 1902 68 FP
BDSTDN, GARY D, 113 DELAWARE SUITE F,66048
682-6826
46 M 2301 74 DRS
BUESING, DLIVER R, 230 NINTH,66048
-
10 M 2501 32 GS
CENAC, MARK T, 500 EISENHDWER RD,66048
727-6000
27 M 401 49 GS
CDMBS, PETER S, 213 DELAWARE,66048
682-0242
14 M 4101 41 IM
DE SDUZA, DERRICK J, 2201 S FDURTH,66048
651-6030
43 M 49501 66 GS
DECENA, IMMACULADA, PD BDX 1675 VAMC,66048
-
45 F 74810 68 EM
DUYSAK, SAMI, 520 6TH AVE,66048
682-6661
22 M 90201 47 IM
FEVURLY, CHRIS D, 500 EISENHDWER,66048
727-6000
54 M 1902 79 IM
GERBER, HARRY A, 605 N SIXTH,66048
-
96 M 5606 30 FP
GRAHAM, KENNETH L, 500 EISENHDWER RD,66048
727-6000
21 M 3840 45 GS
GRAHAM, THDMAS W, 500 EISENHOWER RD,66048
727-6000
26 M 3840 50 IM
GRISDLIA, ANDRES, 424 WALNUT ST,66048
682-5400
27 M 84708 50 DRS
HAMMEKE, JDHN C, 3601 S 4TH ST TRAFFICWAY,66048
682-5201
27 M 401 61 DPH
JDHLER, TERRY HARTWIG, 500 EISENHDWER RD,66048
727-6000
40 M 2802 67 FP
JDHNSDN, PAUL D, 520 SIXTH AVE,66048
682-6661
36 M 1902 61 FP
KAVI, NAGESH G, 3116 LAKEVIEW CL,66048
682-2000
32 M 49509 58 IM
LIPDFF, JAY I, 410 S FDURTH,66048
682-6950
50 M 3519 75 IM
MCCDLLUM, WILLIAM B, 3601 S FDURTH,66048
682-6661
41 M 1902 66 TS
MERRITT, W HENRY, 44 WESTWDDD DR,66048
682-6661
14 M 702 39 GS
MILLS, VERNDN A, 520 SIXTH AVE,66048
682-6661
51 M 1902 77 PD
PARKER, RDBERT W, 500 EISENHOWER RD,66048
727-6000
45 M 1902 71 FP
PRAY, CLAUDIA M, 529 DELAWARE,66048
682-4771
52 F 1902 74 PD
RABE, MELVIN A, 520 SIXTH AVE,66048
-
14 M 1902 37 DD
STRUTZ, WILLIAM C, 68 WESTWDDD DR,66048
682-8868
08 M 5606 43 R
STUMP, HARL G, 4101 S 4TH TRAFFICWAY,66048
682-2000
39 M 1902 65 GS
VDDRHEES, CARRDLL D, 520 SIXTH AVE,66048
682-6661
25 M 1902 52 FP
VDDRHEES, GDRDDN S, 520 SIXTH AVE,66048
642-6661
12 M 1902 39 IM
WALTZ, CHARLES A, 520 SIXTH AVE,66048
682-6661
35 M 3840 62 TS

LEBO—316
(Flint Hills Society)

HUNTER, KENNETH R, ,66856
256-2565
07 M 1902 39 FP

LENORA—913
(Northwest Kansas Society)

STEICHEN, EDWARD F, ,67645
-
05 M 1601 31 FP

LIBERAL—316
(Seward County Society)

ALLEN, RAY E, 2 PLAZA DR,67901
624-5691
37 M 1902 64 IM
CAEDO, CARMELITA D, PD BDX 1643,67901
624-1651
41 F 74801 63 R
CAMPIDN, WDDDRDW M, 121 W THIRD,67901
624-2594
13 M 1902 39 IM
ESTRADA, EDMUNDD C, 1023 N KANSAS SUITE 3,67901
733-1331
43 M 74801 67 GS
ESTRADA, LINA, 1023 N KANSAS SUITE 3,67901
733-1331
43 F 74801 68 PD
GRIMES, I RDSS, 222 W 15TH,67901
624-1676
27 M 3901 54 TS

HARRIS, NORVAN D, BOX 97, 67901
624-3811
20 M 1902 44 08G
HOLCOMB, WILLIAM M, 15 E 11TH,67901
624-2252
31 M 3901 56 GS
KOONS, JESS W, 1210 N WASHINGTON,67901
624-3841
27 M 1902 57 OPH
NEVINS, RICHARD L, PO BOX 1824,67901
624-1841
47 M 3901 73 FP
PROCHAZKA, OTTO F, BOX 1809,67901
-
12 M 1902 38 OO
RATHBUN, EDWIN O, 610 W 11TH,67901
624-1841
36 M 1902 62 FP
REESE, JACK O, 15 E 11TH,67901
624-6226
32 M 1902 57 FP
WADE, THEODORE E, 318 N LINCOLN,67901
354-5275
04 M 512 30 OO
ZAINALI, ASSADOLLAH, 601 LILAC DR,67901
624-1651
46 M 51701 72 R

LINDSBORG—913
(McPherson County Society)

FREDRICKSON, DUANE E, 121 W LINCOLN,67456
227-3371
39 M 1902 66 FP
FULLER, DERYL D, 121 W LINCOLN,67456
227-3371
25 M 1902 50 FP
MURFITT, MALCOLM C, 231 N MAIN,67456
227-2732
13 M 801 41 FP

LYNDON—913
(Franklin County Society)

STOUT, NILES M, ,66451
828-4521
16 M 1902 50 FP

LYONS—316
(Rice County Society)

GRIMES, JAMES T, 1221 W NOBLE,67554
257-5124
27 M 1902 53 FP
SIEMENS, RICHARD A, 1221 W NOBLE,67554
257-5124
30 M 1902 59 FP
TOBIAS, ROGER R, 1017 S BELL,67554
278-2123
M FP
WOLF, CURTIS V, 1221 W NOBLE,67554
257-5124
37 M 1902 64 FP

MADISON—316
(Flint Hills Society)

BROWNING, WILLIAM R, 205 W MAIN,66860
437-2140
44 M 1902 73 FP

MANHATTAN—913
(Riley County Society)

BAKER, RICHARD B, 1133 COLLEGE,66502
537-4200
42 M 4113 68 ORS
BALL, RALPH G, 215 S DELAWARE,66502
-
03 M 1902 27 OO
BAMBARA, JOHN F, PO BOX 128,66502
539-5363
46 M 1902 75 PATH
BARLOW, JOHN M, 1133 COLLEGE,66502
539-3504
45 M 1102 71 OTO
BASCOM, CHARLES H, C/O LAFENE STU HLTH CTR,66506
532-6544
31 M 1902 55 FP
BASCOM, GEORGE S, 1133 COLLEGE,66502
539-6574
27 M 2401 52 GS
BOESE, KENNETH M, 1133 COLLEGE AVE,66502
776-4744
25 M 1902 56 FP
BROWN, ROBERT M, 1133 COLLEGE,66502
537-4940
31 M 1902 63 FP
BURDICK, BRUCE M, 117 S FIFTH,66502
776-9411
25 M 512 53 P
CATHEY, ROBERT H, 1133 COLLEGE AVE,66502
537-4990
42 M 1902 68 O
CRANE, C HERBERT, 1133 COLLEGE,66502
537-9030
22 M 3520 46 PO
DURKEE, WILLIAM R, 1133 COLLEGE AVE,66502
776-4744
23 M 1902 45 IM
FAIRCHILD, JOHN A, 756 COLLEGE HTS CIR,66502
-
14 M 3006 41 OO
FISCHER, REX R, 1133 COLLEGE,66502
776-1400
34 M 3005 60 OBG
FISHER, RONALD M, N CENTRAL GUIDANCE CENTER,66502
234-9566
50 M 3305 76 P
FREEMAN, FRED A, MANHATTAN MED CENTER,66502
537-8710
42 M 1902 69 U
GARDNER, JAMES D, 1133 COLLEGE AVE,66502
537-4940
43 M 2834 71 IM
HANCOCK, DANIEL E, 1133 COLLEGE PO BOX 128,66502
539-5363
45 M 2803 PATH
HAUN, RUDY T, 1133 COLLEGE SUITE 11,66502
776-1400
49 M 1902 78 OBG
HEASTY, ROBERT G, 2030 SCHEU DR,66502
-
11 M 3519 38 OO
HOSTETTER, PHILIP H, 821 POYNTZ,66502
537-2544
17 M 1902 42 FP
HUNTER JR, JAMES S, 1133 COLLEGE,66502
539-5501
18 M 2301 41 OBG
JUBELT, HILBERT P, 1133 COLLEGE,66502
537-9030
19 M 1611 43 PO
KALDOR, RICHARD H, PO BOX 128,66502
539-5363
40 M 2401 66 PATH
KIRK, THOMAS E, 2312 ANDERSON,66502
776-0450
44 M 3005 71 OPH
KLINGLER JR, EUGENE A, 1133 COLLEGE,66502
539-5341
35 M 1902 62 GS
KLOBASA, CHARLES L, GUIDANCE CENTER,66502
539-5337
49 M 2803 75 CHP
LAFENE, BENJAMIN W, 1844 ANDERSON AVE,66502
-
01 M 3806 31 OO
LOWE, STANLEY W, 1133 COLLEGE AVE,66502
776-3451
32 M 1902 59 OPH
LYONS JR, FRANK C, 1133 COLLEGE AVE,66502
539-7641
44 M 3840 70 OR
MARSHALL, RONALD L, 1133 COLLEGE AVE,66502
539-5322
42 M 3005 67 OBG
MARTIN, DANIEL C, LAFENE HEALTH CENTER KSU,66506
532-6544
30 M 1902 58 IM
MCKNIGHT, DAVID E, 1133 COLLEGE AVE,66502
539-7641
32 M 1902 62 R

MCNEIL, ELBERT O, 1133 COLLEGE,66502
537-9030
22 M 702 48 PO
MEEK, PALMER F, 1133 COLLEGE,66502
537-2651
45 M 1902 71 1M
MILLER, ABRAHAM H, 1133 COLLEGE,66502
527-2651
29 M 4101 54 1M
MOSIER, MICHAEL L, 215 SOUTHWIND PL,66502
776-9761
52 M 1902 77 FP
MOSIER, STEVEN J, 215 SOUTHWIND PLACE,66502
776-9761
49 M 1902 74 FP
MOWRY, GERALD L, 1133 COLLEGE,66502
776-1400
26 M 1902 53 OBG
OLNEY, ROBERT D, 1133 COLLEGE AVE,66502
539-7555
27 M 3005 51 GS
PETERSON, JACK T, 1133 COLLEGE,66502
539-5363
25 M 1902 50 PATH
PHILIPP, JOSEPH THEODORE, 1115 WATERS,66502
537-7373
45 M 1902 67 OPH
PHILLIPS, STEPHEN P, 1501 JARVIS,66506
-
17 M 1902 45 OO
REITZ, LELAND C, 1133 COLLEGE,66502
537-2651
36 M 1902 63 1M
REITZ, ROGER P, 1133 COLLEGE,66502
537-2651
32 M 1902 59 1M
ROSE, GRAHAM C, 1133 COLLEGE,66502
537-9030
46 M 4706 70 PO
SHIELDS, THOMAS M, 1133 COLLEGE,66502
539-5341
49 M 1902 74 GPVS
STONE, G REX, 1133 COLLEGE AVE,66502
539-7555
29 M 1902 54 GS
TAYLOR, BARBARA D, 1133 COLLEGE,66502
357-4940
50 F 1902 75 1M
TIEMANN, WILLIAM H, 1133 COLLEGE,66502
537-4940
42 M 3005 67 FP
TOUT, ROBERT C, LAFENE STUDENT HLTH CTR,66506
532-6544
27 M 4812 53 FP
VOLKMANN II, HARLEY W, 1133 COLLEGE AVE,66502
539-7641
47 M 1902 72 R
WALL, KEVIN K, 215 SOUTHWIND PL,66502
776-9761
53 M 2101 79 FP
WHITE, THADDEUS H, 1735 ANDERSON AVE,66502
-
15 M 1902 42 OO

MANKATO—913
(Republic County Society)

KIMBALL, RICHARD R, 102 S CENTER,66956
378-3511
45 M 1001 72 FP

MAPLE HILL — 913
(Flint Hills Society)

KEITH, ROBERT MARSHALL, RURAL ROUTE 1,66507
-
27 M 801 54 P

MARION—316
(Marion County Society)

GUERRA, TOMAS H, 122 S CEDAR,66861
382-2137
33 M 84704 65 1M

HODSON, DON W, 537 S FREEBORN,66861
382-3722
53 M 1902 79 FP

MARYSVILLE—913
(Northeast Kansas Society)

ARGO, DONALD A, 808 N 19TH,66508
562-2303
36 M 3005 64 FP
LAWS, LEWIS R, 808 N 19TH,66508
562-2303
25 M 1902 54 FP

MAYETTA — 913
(Shawnee County Society)

BEATY, JAMES R, RT 2 BOX 11,66509
354-1111
32 M 5101 66 EM

McLOUTH—913
(Jefferson County)

SNOOK, ROBERT RUFUS, ,66054
796-6116
11 M 1902 42 FP

McPHERSON—316
(McPherson County Society)

BILLINGS, THOMAS, 400 WEST 4TH,67460
241-5500
39 M 1902 66 FP
BRANDSTED, ERNEST C, 400 W 4TH,67460
241-1654
18 M 1606 44 OBG
CLASSEN, SAMUEL O, 400 WEST FOURTH,67460
241-7033
53 M 1902 78 1M
COLLIER, WILLIAM J, 400 W 4TH,67460
241-1766
25 M 3605 49 TS
DYCK, ARTHUR H, C/O PEOPLES BANK/TRUST,67460
241-0357
03 M 1902 28 FP
FERREE, RICHARD ALLAN, 400 W FOURTH,67460
241-7400
51 M 3006 76 FP
FIELDS, GALEN W, 333 C - S LAKESIDE DR,67460
-
15 M 1902 49 OO
JOHNSON, J RICHARD, 400 W 4TH,67460
241-4293
28 M 1902 55 1M
PIERSON, WEIR, BOX 1028,67460
241-1445
17 M 1902 44 FP
PRICE, VAUGHAN C, PO BOX 451,67460
-
05 M 4706 29 GS
SCHURLE, DALE R, 400 W FOURTH,67460
241-7033
53 M 1902 78 1M
THOMAS, GREGORY MCQUEEN, 400 W FOURTH,67460
241-7400
47 M 1902 73 FP

MEADE—316
(Iroquois County Society)

HILL, RICHARD H, 234 EAST CARTHAGE,67864
873-2113
18 M 1902 44 FP

MEDICINE LODGE—316
(Tri-County Society)

HOFFER, JOHN G, 910 N WALNUT,67104
886-3222
13 M 1902 44 GS

STUCKY, DEAN E, 901 N WALNUT,67104
886-5653
33 M 1902 60 FP

MINNEAPOLIS—913
(Saline County Society)

BARKER, STEVEN E, 311 N MILL,67467
392-2144
51 M 1902 76 FP
WEDEL, KENNETH D, 311 N MILL ST,67467
392-2144
32 M 1902 60 FP
WEDEL, KERMIT G, 311 N MILL ST,67467
392-2144
32 M 1902 60 FP

MINNEOLA—316
(Iroquois County Society)

STEPHENS, CHARLES, MINNEOLA CLINIC,67865
885-4202
33 M 2803 58 FP

MONTEZUMA—316
(Ford County Society)

CURA, ELSA C, MONTEZUMA CLINIC BOX 384,67867
846-2251
37 F 74808 61 IM

MORAN—316
(Allen County Society)

NEVITT, J RUSSELL, P O BOX 430,66755
-
99 M 3510 32 OO

MOUNDRIDGE—316
(McPherson County Society)

KAUFMAN, WILLARD E, 115 N CHRISTIAN AVE,67107
345-6322
28 M 1902 53 FP
LOGANBILL, VARDEN J, 115 N CHRISTIAN AVE,67107
345-6322
26 M 1902 54 FP

MULVANE—316
(Sedgwick County Society)

COBB, LESLIE H, 102 E MAIN,67110
777-1441
17 M 4804 47 FP

NASHVILLE—316
(Ninnescah Society)

WAYLAN, THORNTON L, ,67112
-
06 M 1902 35 OO

NEODESHA—316
(Southeast Kansas Society)

CHRONISTER, BERT, PO BOX 118,66757
325-2622
38 M 1902 64 FP
MOORHEAD JR, F ALLEN, 709 MAIN ST,66757
325-2200
39 M 1902 65 FP

NESS CITY—913
(Central Kansas Society)

PRAKALAPAKORN, DARANEE, 412 N TOPEKA,67560
798-2233
47 F 89101 69 PD

PRAKALAPAKORN, YANYONG, 412 N TOPEKA,67560
798-2233
43 M 89101 69 GS

NEWTON—316
(Harvey County Society)

ALLEN, FRANCES A, 1112 BOYD,67114
-
15 F 1902 43 OO
BATES, MICHAEL NICHOLS, 126 S PINE,67114
283-4153
50 M 1902 75 OBG
BENTON, JAY S, 301 MAIN,67114
283-3600
23 M 4804 49 OBG
BOLT, MICHAEL, AXTELL CLINIC,67114
283-2800
51 M 1902 76 GS
CAMPBELL, FRANCES S, 1901 E FIRST,67114
283-2400
35 F 4101 61 P
CARPER, IVAN H, 203 E BROADWAY,67114
283-2800
28 M 1902 59 GS
CARPER, OWEN E, AXTELL CLINIC,67114
283-2800
37 M 1902 64 FP
CLAASSEN, MILTON A, 201 S PINE ST,67114
283-3600
32 M 1902 58 ORS
CRAIG, CHARLES C, AXTELL CLINIC,67114
283-2800
45 M 1902 71 ORS
CRANSTON, STEPHEN D, 215 S PINE,67114
283-0027
45 M 1902 71 GS
DYCK, GEORGE, PRAIRIE VIEW INC,67114
283-2400
37 M 6201 64 P
ENNS, EUGENE K, 6 INDIAN LANE,67114
-
15 M 1902 40 OO
FENT, LEE S, 316 OAK,67114
283-0505
14 M 2834 43 GS
FRANSEN, HERBERT, 215 S PINE,67114
283-0033
32 M 6501 60 GS
FRANSEN, PAUL H, BOX 848,67114
283-5040
46 M 6501 71 FP
GLOVER, RICHARD M, AXTELL CLINIC,67114
283-2800
21 M 1902 53 FP
GRABER, CHARLES, 215 S PINE,67114
283-0033
44 M 3006 75 GS
GRISWOLD, DALE G, AXTELL CLINIC,67114
283-2800
27 M 1902 53 IM
GROVE, JOHN A, 407 W 16TH,67114
-
08 M 1606 37 ORS
HENDRICKSON, JON R, AXTELL CLINIC,67114
283-2800
51 M 1902 73 PD
HENDRICKSON, KATHRYN D, AXTELL CLINIC,67114
283-2800
52 F 1902 77 PD
HWA, EUGENE C, 500 MAIN,67114
283-1160
21 M 24216 47 R
IRWIN, RICHARD L, 218 S KANSAS AVE,67114
283-1400
48 M 1902 75 OPH
ISAAC, CHARLES A, 203 E BROADWAY,67114
283-2800
25 M 1902 49 U
KLIEWER, VERNON L, PRAIRIE VIEW MHC,67114
283-2400
31 M 1606 57 CP
KUMAR, SURINDER, 201 S PINE,67114
835-2241
46 M 1902 69 OBG
LINDHOLM, GERALD R, AXTELL CLINIC,67114
283-2800
51 M 1902 76 FP
NACHTIGALL, ANDREW, BETHEL CLINIC,67114
283-3600
28 M 1902 59 PD

O'TOOLE, JAMES K, PRAIRIE VIEW HOSPITAL,67114
283-2400
28 M 1643 54 P
OLSON, ERWIN T, BETHEL CLINIC,67114
283-3600
19 M 1902 47 PO
PREHEIM, DELBERT V, BOX 848,67114
283-5040
13 M 702 42 IM
PRENTISS, HAROLD, 1305 TERRACE DR,67114
283-9433
36 M 1720 62 R
QAMAR, YUSUF, 203 E BROADWAY,67114
283-2800
38 M 70409 62 IM
RADOVANOV, RADMILA, BOX 364,67114
34 F 95702 60 R
RICH, ELDON S, BETHEL CLINIC,67114
16 M 1902 46 OO
SCHMIDT, HERBERT R, CEDAR VILLAGE,67114
03 M 1902 34 OO
SILLS, CHARLES T, 1631 HILLCREST,67114
09 M 1902 37 OO
SIMMONS, ROBERT EARLE, BOX 848,67114
283-5040
49 M 1902 74 IM
TANDOC JR, VALENTIN T, BETHEL CLINIC,67114
283-3600
39 M 74809 62 U
TOMPKINS, CARL O, 316 OAK STREET,67114
283-1380
22 M 1902 51 FP
VAUGHAN, DONNA A, 201 S PINE,67114
283-3600
45 F 1902 71 IM
VOGT, VERNON W, BETHEL CLINIC,67114
283-3600
22 M 3005 53 FP
VONGKASEMSIRI, SUNAN, 1709 CYPRESS LN,67114
283-1160
41 M 89101 67 R
WEBER, ROY R, 215 S PINE,67114
283-3025
46 M 1902 73 IM
WHEELER, DWIGHT E, BETHEL CLINIC,67114
283-3600
50 M 2012 76 IM
WIENS, J WENDELL, 201 S PINE,67114
283-3600
32 M 1902 59 GS

NORTON—913
(Northwest Kansas Society)

COLIP, F MERLYNN, 711 N NORTON,67654
877-3305
35 M 1902 61 FP
COOPER, ARTHUR E, 305 W WILBERFORCE,67654
08 M 1611 34 GP
HARTLEY, ROY WILEY, 711 N NORTON,67654
877-3305
37 M 1902 63 FP
HARTMAN, ROGER L, 711 N NORTON,67654
877-3305
35 M 1902 61 FP
LONG, ROBERT C, 711 N NORTON,67654
927-3305
27 M 1902 53 GS

NORTONVILLE—913
(Jefferson County)

MADISON, WILLARD A, ,66060
886-2110
20 M 1902 51 FP

OAKLEY—913
(Northwest Kansas Society)

OHMART, RICHARD V, PO BOX 756,67748
672-3262
36 M 1902 62 FP
SEKAVEC, GORDON B, 209 CENTER AVE,67748
672-3351
07 M 1902 38 FP

OBERLIN—913
(Northwest Kansas Society)

PALMER, MARGUERITE L, BOX 110,67749
475-2221
25 F 5404 54 GP
SIMPSON, ROBERT LIMBAUGH, 902 W COLUMBIA,67749
475-2221
25 M 4706 51 GS
WHITAKER, REN R, 902 W COLUMBIA,67749
475-2222
37 M 5404 66 FP

OLATHE—913
(Johnson County Society)

ARONOFF, MICHAEL E, 407 S CLAIRBORNE STE 209,66062
782-3953
39 M 1604 64 ENT
ASBURY, LAWRENCE J, OLATHE COMMUNITY HOSP,66062
782-1451
45 M 2878 77 EM
BEEBE, EDMER, 420 EAST CEDAR,66061
03 M 5605 32 FP
BLISS, JOY V, 42 HOLLY DRIVE,66062
782-2292
42 F 3005 68 ANES
BLUM, MICHAEL A, D.O., 401 S CLAIRBORNE,66062
764-7060
47 M 2878 73 PD
BROWN, PAUL W, 405 S CLAIRBORNE,66062
782-3322
44 M 1902 70 FP
CHENOWETH, JOHN R, D.O., 1003 STRATFORD RD,66062
36 M 2878 70 OST
DELPHIA, ROBERT E, 401 S CLAIRBORNE,66062
782-1610
24 M 1902 56 FP
EIDT, DAVID W, 407 S CLAIRBORNE,66062
782-8487
44 M 2501 70 FP
EIDT, LAURENCE A, 407 SOUTH CLAIRBORNE,66062
782-8487
44 M 1902 71 FP
FORTUNE, CEDRIC B, PO BOX 910, 66061
782-3322
40 M 1902 66 FP
GLAZZARD, CHARLES D, 407 S CLAIRBORNE,66062
782-3384
28 M 2507 56 P
HALVORSON, HOWARD C, 407 S CLAIRBORNE,66062
782-2020
41 M 5404 66 U
HUDSON, ROBERT P, 12925 FRONTIER RD,66061
588-7040
26 M 1902 52 IM
JENSEN, THOMAS M, 407 S CLAIRBORNE,66062
782-1148
47 M 3005 73 ORS
LAIRD, DALE O, ONE PATRONS PLAZA,66061
782-3631
42 M 1902 68 OPH
MATHEWS, MICHAEL WM, 114 S CLAIRBORNE,66062
764-9075
53 M 2803 79 FP
MATHEWS, ROBERT C, 300 S ROGERS RD,66062
782-1451
50 M 2834 77 EM
MATTHEW, WILLIAM L, 405 S CLAIRBORNE,66062
782-3322
29 M 1902 56 FP
MCCANN, WILLIAM E, PO BOX 8,66061
782-0262
22 M 3901 48 FP
MEE, ADRIAN W, 28 HOLLY DRIVE,66062
782-2292
19 M 1902 54 ANES
MELENDRES, JUANITO M, 7400 GLEASON RD - RR 1,66061
684-4649
36 M 74809 61 PD
MENDLICK, R MICHAEL, 407 S CLAIRBORNE STE 101,66062
782-1148
44 M 1902 70 ORS

MORGAN 11, OAV1O LLOYO, 807 S CLA1RBONRE,66062
782-8300
49 M 2820 77 1M
NOTT1NGHAM, ROBERT M, 401 CLA1RBORNE,66062
782-1610
49 M 1902 78 FR
RIERRON, GEORGE J, 540 EAST SANTA FE,66061
782-0260
22 M 1902 47 FP
ROMONOO, STEVEN A, 300 SOUTH ROGERS RO,66062
782-2292
47 M 1902 73 ANES
RUHLEN, JAMES L, 807 S CLA1RBORNE,66062
782-8300
46 M 1902 72 1M
SEAMAN, LAUREN 1, 1613 E SHER1OAN,66062
-
07 M 6001 38 FP
SETTLE JR, RUSSELL O, 407 S CLA1RBORNE,66062
782-3384
35 M 1902 60 P
SHANKER, STUART G, 401 S CLA1RBORNE STE 200,66062
764-7060
49 M 2803 75 RO
SHEFFER, KE1TH O, 407 S CLA1RBORNE STE 101,66062
782-1148
37 M 1720 67 ORS
SNYOER JR, RICHARO HENRY, 300 S ROGERS ROAO,66061
782-1451
45 M 1902 73 ANES
ST1TES 111, HAROLD W, 807 CLA1RBORNE,66062
782-8300
50 M 2803 80 IM
V1LLANUEVA, CESAR L, 401 S CLAIRBORNE,66602
764-4467
39 M 74802 65 OBG
W1LL1AMS, THOMAS A, 1948 E SANTA FE,66062
764-4800
36 M 1902 62 FP
YEOMANS, RONALO N, 405 S CLAIRBORNE SU1TE 4,66062
782-3073
40 M 1902 67 OBG

ONAGA—913
(Pottawatomie County Society)

FLECKENSTE1N, CHARLES S, 501 LUCIEN ST,66521
-
07 M 1902 36 OO
WALSH, THOMAS E, ONAGA CL1NIC,66521
889-4241
48 M 1902 74 FP

OSAGE CITY—913
(Flint Hills Society)

AOAMS, OWIGHT, 603 HOLL1OAY,66523
528-3165
M 1902 GP
W1LL1AMS, HOMER J, 611 S S1XTH,66523
-
05 M 1902 31 FP

OSAWATOMIE—913
(Miami County Society)

APPENFELLER, W1LL1AM O, 524 BROWN AVE,66064
755-3166
25 M 1902 53 FP

OSWEGO—316
(Labette County Society)

BURGESS, ARTHUR P, 504 5TH STREET,67356
795-4427
19 M 1902 52 FP

OTTAWA—913
(Franklin County Society)

BLANKENSHIP, JIM O, 1320 S ASH,66067
242-5581
50 M 2834 76 FR
CORDER, S SCOTT, 1502 S CEDAR,66067
242-2641
51 M 1902 76 FP
GOLLIER, ROBERT A, *,66067
-
13 M 1902 37 OO
GOLL1ER 11, ROBERT A, 1320 S ASH,66067
242-1620
40 M 1902 66 FR
HAOLEY, OELMONT C, 1320 SOUTH ASH,66067
242-3891
35 M 1902 64 FR
HENN1NG, CALV1N W, 1502 CEOAR,66067
-
05 M 1902 35 FP
LAURY, OAV1O G, 1320 S ASH,66067
242-1620
17 M 1606 44 FP
RHILGREEN, OONALO E, 1320 S ASH,66067
242-3891
39 M 1602 67 FP
RANSOM, W1LLARO B, 1320 S ASH,66067
242-1620
49 M 1902 77 FP
REYES JR, FRANC1SCO A, 1320 S ASH,66067
242-5312
38 M 74801 61 GS
SREER, LOU1S N, PO BOX O,66067
242-1257
14 M 1606 41 FR
STREHLOW, CHESTER H, RROFESSIONAL PLAZA BLOG,66067
242-3891
30 M 1902 57 FR

OVERBROOK—913
(Shawnee County Society)

RUBLF JR, JAMES L, OVERBROOK COMM CL1N1C,66524
665-2205
26 M 1902 53 FR

PAOLA—913
(Miami County Society)

BANKS, ROBERT E, RO BOX 298,66071
294-2305
29 M 1902 55 FR
ROWLETT, JACK G, RO ORAWER A,66071
294-2356
21 M 1902 52 FP
STANLEY, REX C, PO ORAWER A,66071
294-2056
24 M 1902 52 GS

PARSONS—316
(Labette County Society)

AVES, AGNES, 1509 MA1N,67357
421-0600
38 F 74801 59 1M
AVES, RENATO B, 1509 MAIN STREET,67357
421-0600
35 M 74801 59 GS
BEATY, EVERT C, 2601 GABRIEL,67357
421-6550
18 M 1902 44 GP
CAING, MAT1LDE V, 2601 GABRIEL,67357
421-6550
41 F 74801 63 PO
CAREY, LARRY J, LABETTE CO MEO CL SU1TE 5,67357
421-8361
51 M 1902 74 FP
CRAMER, GUY W, 412 MURDOCK,67357
-
11 M 1902 39 OO
OA1Z, ANTONIO S, LABETTE COUNTY MEO CTR,67357
421-4880
37 M 74810 63 OR
OILLON, W1LLIAM L, LABETTE CO MEO CL BOX H,67357
421-0881
45 M 1902 71 ORS
HENDERSON, CHARLES F, 1617 GRAND,67357
-
14 M 1902 40 OO
K1SHORE, ROY N, LABETTE CLINIC #3,67357
421-4062
44 M 49511 66 OTO

KISHORE, SHEELA, 3604 GABRIEL #222,67357
421-2741
43 F 49511 66 ANES
LAVA, CHIRUND, PO BOX 290,67357
421-6210
40 M 89102 63 GS
MARTIN, EARL A, 1516 GRAND,67357
-
07 M 1606 35 OO
MILLER, CHARLES H, 2819 CLARK,67357
-
07 M 3006 32 OO
MILLER, DEAN M, 203 CRESTVIEW,67357
421-4880
22 M 1902 48 R
MILLER, STEPHEN FRANCIS, 1509 MAIN,67357
421-0600
45 M 1902 70 GS
PACE, JOHN D, KATY HOSPITAL CLINIC,67357
-
94 M 1902 20 FP
PAI, RADHA V, PO BOX 1057,67357
421-0080
45 F 6701 ANES
PAI, VARADARAJ S, PO BOX 1057,67357
421-0080
42 M 6701 U
PARANJOTHI, SUBRAMONIAM P, 1509 MAIN,67357
421-6160
39 M 49531 64 IM
PAULS, DANIEL N, PO BOX 1014,67357
421-1431
45 M 1902 71 IM
ROTHSTEIN, TERRY B, 220 NORTH 32ND,67357
421-5900
43 M 1606 69 OPH
SHARMA, ARUN L, 1509 MAIN,67357
421-0600
46 F 49503 69 FP
TANGCHUPONG, CHANTRASIRI, PO BOX 1054,67357
421-2460
47 F 89104 71 PD
TANGCHUPONG, SAROHD, PO BOX 1054,67357
421-2460
43 M 89102 69 OBG
VERMA, ASHA, 400 KATY,67357
421-9090
37 F 49530 63 PD
VICHYANOND, PAKIT, LABETTE CO MEDICAL CENTER,67357
421-2460
58 M 89101 75 PD
WHITE, JOHN P, PARSONS CLINIC,67357
421-0600
17 M 1902 42 FP

PEABODY — 316
(Harvey County Society)

MARTINEZ, KAY C, 500 W FOURTH,66866
983-2131
49 F 1902 77 FP

PHILLIPSBURG—913
(Central Kansas Society)

RICHARDS, JON F, 205 W STATE,67661
-
50 M 1902 75 FP

PITTSBURG—316
(Crawford County Society)

ARMSTRONG, HAROLD J, PROFESSIONAL BUILDING,66762
232-2600
40 M 1902 68 ORS
BENA, JAMES H, 109 EAST 9TH,66762
231-6950
12 M 3005 36 PD
BERKEY, VERNON A, KIRKWOOD BLDG,66762
231-7650
18 M 1902 43 R
BIERLEIN, KENNETH J, 812 S CATALPA,66762
-
06 M 1606 33 OO
COOMER, TYLER E, 315 NATL BANK BLOG,66762
231-7730
30 M 2101 59 GS
COOPER, KENT J, 909 CENTENNIAL,66762
231-6280
41 M 1902 73 FP
ERICKSON, CLARENCE W, 217 NATL BANK BUILDING,66762
231-7400
06 M 1902 33 IM
ESCH, JOHN G, 207 KIRKWOOD BLDG,66762
231-5360
24 M 3006 48 GS
FREEMAN, MALCOLM C, 1102 VILLAGE DR,66762
231-6100
44 M 35207 68 ANES
GOMETZ, MODESTO S, 909 E CENTENNIAL #6,66762
231-2490
35 M 72601 63 PD
HOLSINGER, DONALD M, 1015 MT CARMEL PL,66762
231-5900
38 M 1902 64 IM
HUEBNER, ROBERT STEPHAN, NATIONAL BANK BLDG,66762
231-6160
42 M 1606 67 GS
HUERTER, DAVID F, 909 CENTENNIAL,66762
231-1650
46 M 1902 72 IM
LANCE, RAYMOND W, 608 W QUINCY,66762
-
22 M 1902 47 OO
LEFFLER, PAUL B, 309 WINWOOD,66762
-
02 M 1902 40 OO
MILLER, EARL E, 1312 S BROADWAY,66762
231-6410
13 M 1902 37 ENT
MULLER, SAMUEL B, 611 W QUINCY,66762
-
05 M 1902 34 OO
NEWMAN, CLIFFORD B, 1204 E 7TH,66762
-
01 M 1902 28 OO
OGERS, RODNEY K, 909 CENTENNIAL,66762
231-4300
M 1902 74 IM
PAPP JR, S DEAN, R 5 BOX 293,66762
231-7650
46 M 1902 72 DR
PARSI, MANUTCHEHR K, 909 CENTENNIAL,66762
231-3770
38 M 51701 64 OBG
POGSON, GEORGE W, 1015 MT CARMEL PLACE,66762
231-5900
24 M 1902 47 IM
RAMIREZ, AUGUSTO H, 909 CENTENNIAL,66762
231-1600
32 M 26407 58 IM
RAMIREZ, IRENE, 909 CENTENNIAL,66762
231-6280
F PD
SCHLEMMER, ROGER B, 1009 S BROADWAY,66762
231-6380
37 M 1902 68 OPH
SEGLIE, FLOYD RONALD, 909 CENTENNIAL DR SUITE 3,66762
231-6280
43 M 1902 69 FP
TWEET, FREDRICK A, RR 5 BOX 196,66762
231-6100
39 M 1602 66 PATH
WOOD, DOUGLAS H, 413 W JEFFERSON,66762
-
11 M 5605 36 OO
YAGHMOUR, TALAAT E, 905 NORTH LOCUST,66762
231-0850
40 M 33002 64 U
ZABEL, KENNETH P, 909 CENTENNIAL,66762
231-1650
37 M 1902 65 IM

PLAINVILLE—913
(Central Kansas Society)

KELLERMAN, RICK, 409 S COCHRAN,67663
434-4609
M 1902 FP
PAGE, O VALE, 409 S COCHRAN ST,67663
434-4609
20 M 1902 51 FP
PEDERSON, ARNOLD M, 409 S COCHRAN,67663
434-4609
22 M 1902 51 FP

PLEASANTON—913
(Bourbon County Society)

JUSTUS, WILLIAM J, ,66075
352-6134
29 M 1902 55 FP

PRATT—316
(Ninnescah Society)

AM8LER, CARL O, 200 COMMOOORE,67124
672-6476
31 M 1902 57 R
BARKER, PATRICK N, 420 COUNTRY CLU8 RO,67124
672-7411
45 M 1902 71 GS
BLACK, CYRIL V, 223 E 4TH,67124
672-6403
05 M 4802 30 GS
CHAFFIN JR, GOOOLOE S, 420 COUNTRY CLUB RO,67124
672-7411
50 M 4705 75 FP
OILLON, STEVEN C, 602 E SECONO,67124
672-5558
53 M 1902 78 1M
FILLEY, VERNON W, 310 E 2NO,67124
672-5555
13 M 3005 43 GS
FREEMAN, F GILES, 310 E 2NO,67124
672-5555
18 M 1902 44 FP
GARO, RAYMONO F, 593 TERRACE OR,67124
-
01 M 1902 27 GS
PITMAN, WILL O, 717 WEST 3RO,67124
-
98 M 1902 25 OO
OUENZER, RONALO W, 420 COUNTRY CLU8 RO,67124
672-7411
46 M 1601 73 1M
S18ALA, JUSTO L, 200 COMMOOORE,67124
672-7159
20 M 74802 49 R
SUITER, OANIEL JAY, 420 COUNTRY CLUB,67124
672-7411
44 M 1902 71 GE
THORPE, FRANCIS A, 2 LAKE ROAO,67124
672-5555
08 M 1606 35 FP
WARO, ROBERT L, 420 COUNTRY CLUB,67124
672-7411
24 M 1902 52 FP
WOLFF, FREOERICK P, 223 E 4TH,67124
672-6403
20 M 1902 44 1M

PROTECTION—316
(Iroquois County Society)

GLENN, LYLE G, 146 BROAOWAY BOX 447,67127
622-4686
12 M 1606 40 FP

QUINTER—913
(Northwest Kansas Society)

GUNTER, CARL C, QUINTER CLINIC 8LOG,67752
754-3333
20 M 1902 51 FP
HIESTERMAN, HERMAN W, QUINTER CLINIC 8LOG,67752
754-3333
23 M 1902 51 FP

RANSOM—913
(Central Kansas Society)

MCLAIN, KENNETH, BOX 247,67572
731-2295
21 M 1902 46 FP

RUSSELL—913
(Central Kansas Society)

ELOREOGE, LOUIS O, 222 S KANSAS,67665
483-6000
48 M 74809 79 GP
MERKEL, EARL O, SHIELOS BLOG,67665
483-2178
32 M 1902 57 FP
PANICHA8HONGSE, SAM8HUNOH, 213 WEST 7TH,67665
483-2178
43 M 89101 67 GS
PETTIJOHN, WALTER J, 624 W 12TH,67665
-
12 M 1902 37 OO
STARKEY, JERALO L, 326 MAIN,67665
275-9444
30 M 1902 56 FP
SWANN, CLAIR L, 112 W 7TH,67665
483-4212
13 M 1902 39 1M
WHITE, FAGAN N, 356 W 5TH,67665
-
11 M 702 36 OO

SABETHA—913
(Northeast Kansas Society)

KENNALLY, KEVIN P, 1115 MAIN,66534
284-2141
53 M 1902 78 FP
MONTGOMERY, THOMAS ALLEN, 1013 WYOMING,66534
-
10 M 1902 49 OO
WENGER, GREGG O, 1115 MAIN,66534
284-2141
M 1902 78 PO
YULICH, JOHN O, PO 80X 227,66534
284-2125
33 M 1902 59 FP

SALINA—913
(Saline County Society)

ALLEN, MONTE L, 600 S SANTA FE,67401
827-0307
36 M 1902 61 OTO
ANOERSON, JOOY, 737 E CRAWFORO,67401
827-7261
32 F 1902 59 IM
BAXTER, W REESE, P O 80X 1707,67401
825-8221
47 M 1902 73 FP
BROWN, ROBERT WAYNE, PO BOX 1747,67402
825-7251
23 M 1902 55 1M
8RUMMETT, RICHARO R, P O BOX 1707,67401
825-8221
34 M 1902 64 FP
8RUNGAROT, BERNARO A, 400 E 8ELOIT,67401
-
21 M 3006 46 OO
CLARK, OAVIO H, 617 E ELM,67401
825-8221
36 M 1902 62 FP
COFFEY, ROY B, 671 FLMORE ORIVE,67401
823-6397
24 M 1902 47 ORS
CONNELLY, MAURICE R, RR 3 BOX 44,67401
-
12 M 2002 38 OO
CONNER, 8RIAN, 1518 B EAST IRON,67401
825-2272
46 M 1902 OPH
COSSETTE, JERROLO E, 909 E WAYNE,67401
823-7225
46 M 1902 75 ENT
CULTRON, FRANK T, 800 E CRAWFORO,67401
823-8151
10 M 1643 38 OPH
O'SOUZA, BISMARCK C, 116-A S 7TH,67401
827-9526
45 M 49501 67 R
OOWELL, JAMES C, 645 E IRON,67401
827-7255
26 M 1611 49 IM
ORAEMEL, H RICHARO, 600 S SANTA FE ST,67401
827-0307
18 M 1902 53 OTO

OREHER, HENRY S, 737 E CRAWFORD,67401
827-7261
18 M 1902 43 IM
EATON, GLEN E, RR 6 BOX 359,67401
827-3064
28 M 1902 54 ANES
EATON, LESLIE F, RR 1 BOX 346,67401
-
06 M 1902 32 OO
ELLISON, PAUL O, 1499 E IRON,67401
825-7271
35 M 2105 60 OPH
FEIGHNY, ROBERT E, 2437 VILLAGE,67401
-
20 M 1902 51 ORS
FORSTER JR, LOUIS G, BOX 1747 - 130 W CLAFLIN,67401
825-8221
47 M 1902 73 FP
FRANCIS, ANTHONY E, 519 S SANTA FE,67401
827-4429
54 M 1902 77 ORS
FREEMAN, RAYMOND S, 737 E CRAWFORD,67401
827-7261
20 M 702 50 PO
GANS, FREDERICK A, 950 S ELEVENTH,67401
-
22 M 2834 46 OO
GRIFFITH, FRANK H, 1493 E IRON,67401
827-0488
45 M 4813 75 OPH
GUNN, MARVIN R, 116A S 7TH PO BOX 1059,67401
827-9526
28 M 3901 54 R
GUZMAN, MANUEL, CKMHC,67401
823-6322
27 M 64901 54 P
HARBIN, GARY LYNN, 523 S SANTA FE,67401
823-7213
50 M 1902 75 ORS
HARRIS, NORMAN R, 430 S 7TH,67401
825-8191
30 M 1902 59 OBG
HASSLER, RANDY O, 645 E IRON,67401
827-9635
45 M 1902 71 U
HATTON, LLOYD W, 709 HIGHLAND,67401
-
06 M 1902 33 P
HERRMAN, ADAM L, 519 S SANTA FE,67401
827-4424
48 M 1902 74 ORS
HESSE, FREDERICK J, PO BOX #918,67401
827-9631
50 M 1902 75 IM
HODGES, MERLE A, 430 S 7TH ST,67401
825-8191
34 M 1902 58 OBG
HOLMAN, JON B, PO BOX 61,67401
827-9366
33 M 1902 63 P
HUNNINGHAKE, RONALD, 617 E ELM BOX 1707,67401
825-8221
51 M 1902 76 FP
JACKSON JR, OELMAS A, 645 E IRON,67401
827-7255
35 M 2101 60 IM
KREHBIEL, MARK A, 617 E ELM,67401
825-8221
49 M 1902 74 FP
KRUCKEMYER, ALAN L, 645 E IRON,67401
823-2215
45 M 1103 71 ORS
LAKE, MAX S, 124 S OAKDALE,67401
-
19 M 3005 43 OO
LASLEY, DAVID A, 645 E IRON,67401
827-9635
22 M 1606 47 U
LIVINGSTON, CHARLES E, 400 E IRON,67401
827-9631
32 M 1611 57 GS
LUNGSTRUM, JACK E, 645 E IRON,67401
823-2215
21 M 1902 59 ORS
MACY, NORMAN E, BOX 1285,67401
827-4053
35 M 1902 60 PATH
MACY, TED L, PO BOX 360,67401
827-7261
43 M 1902 71 GS
MARCHBANKS, DONALD L, 520 COUNTRY CLUB RD,67401
823-2380
24 M 1902 51 FP

MARSHALL, GEORGE W, PO BOX 1705,67401
225-3191
44 M 1902 70 OBG
MARTIN, OLIVER L, 715 E REPUBLIC,67401
827-9631
08 M 1902 37 OBG
MATHIS, JERRY L, 1112 ALBERT,67401
827-9343
35 M 1902 62 POA
MAXWELL, GORDON E, PO BOX 918,67401
827-9631
29 M 1902 55 OBG
MCCRAE, SPENCER C, 519 S SANTA FE,67401
-
18 M 3509 43 OO
MILLER, ELDEN V, 1928 RIDGELEA,67401
827-3061
19 M 1902 44 ANES
MITCHELL, JOHN C, PO BOX 922,67401
827-3061
13 M 1902 38 GS
MOWERY, WILLIAM E, 737 E CRAWFORD,67401
823-6455
23 M 1902 47 GS
NICKELL, WENDELL K, 400 E IRON,67401
827-9618
26 M 1606 50 TS
NIXON, RICHARD R, 153 CHANNEL DR,67401
827-9526
32 M 1643 57 R
NULL, WILLIAM G, PO BOX 918,67401
827-9631
31 M 102 57 PO
OBANDO, GUILLERMO, 2110 KNOLLCREST DR,67401
823-6219
35 M 26404 62 R
PALMER, GERALD K, PO BOX 1285,67401
823-7201
24 M 1803 53 PATH
PETERSON, JAMES E, 116A S SEVENTH BOX 1059,67401
827-9526
53 M 1902 78 OR
REECE, RICHARD J, 116A S 7TH,67401
827-9526
23 M 1902 49 R
RODERICK, JAMES E, 645 E IRON,67401
827-9635
23 M 1902 47 U
ROMEISER, REX S, 645 E IRON,67401
827-9635
41 M 1902 67 U
RUEB, ANDREW E, 11 CRESTVIEW DR,67401
827-6691
11 M 1606 35 GS
SCANLAN, TIMOTHY M, PO BOX 1747,67402
825-7251
46 M 2604 71 FP
SCHMIDT, RAMON WARNER, 400 E IRON,67401
827-9618
39 M 1902 65 GS
SCOTT, CHESTER E, 519 S SANTA FE,67401
827-5549
23 M 1902 51 FP
SEBREE, STEVEN G, PO BOX 360,67401
827-7261
47 M 1902 73 OBG
SLOO, MILO G, 645 E IRON,67401
823-2215
41 M 1902 67 ORS
SMITH, BOYD E, BOX 128S,67401
827-4053
46 M 3005 72 PATH
SMITH, DAVID E, 2106 APPLEWOOD,67401
588-6100
50 M 1902 76 GS
SMITH, HAROLD R, PO BOX 360,67401
827-7261
19 M 1902 51 GS
SNYDER, THOMAS E, 737 E CRAWFORD,67401
827-7261
47 M 1902 OBG
STOSKOPF, LAWRENCE E, 2413 EDGEHILL,67401
823-9498
39 M 1902 72 ANES
TAYLOR, THOMAS F, 430 S OHIO,67401
827-0346
26 M 1902 53 FP
UNAL, ABDURRAHMAN, 116A S SEVENTH BOX 1059,67401
827-9526
49 M 90205 74 TR

WAGENBLAST, HOWARD R, 737 E CRAWFORD,67401
827-7261
21 M 1902 49 FP
WATERS, CLARENCE N, 530 S 5TH,67401
823-6497
13 M 2834 48 O
WEBER, ROBERT W, 645 E IRON,67401
827-7255
26 M 1902 49 IM
WOERNER, DAVID R, 539 S SANTA FE,67401
825-9397
49 M 64914 75 FP

SATANTA—316
(Southwest Kansas Society)

JABEL, JUVENAL T, SATANTA CLINIC,67870
649-2771
43 M 74809 IM
TAOURAN, VIRGILIO, SATANTA CLINIC,67870
649-2771
43 M 74810 43 PATH

SCOTT CITY—316
(Southwest Kansas Society)

DUNN, DANIEL R, 202 COLLEGE,67871
872-2187
49 M 1902 74 FP
HOPKINS JR, B MORRISON, 202 COLLEGE,67871
872-2187
23 M 1902 53 FP

SEDAN—316
(Southeast Kansas Society)

TAYLOR, ELMER W, 120 WEST OSAGE,67361
725-3141
28 M 512 57 FP
WALKER, WILLIAM K, 111 E CHEROKEE,67361
725-3171
18 M 1902 45 FP

SENECA—913
(Northeast Kansas Society)

BERKLEY, NORMAN W, 15 SOUTH 5TH ST,66538
336-2128
31 M 1902 63 FP
GILBERT, J HOWARD, 211 S 4TH, 66538
-
05 M 1902 41 OO
LUEGER DO, JAMES JOHN, 201 N SIXTH,66538
336-6113
51 M 2878 78 GP
MC GEENEY, TERRY L, 201 N SIXTH,66538
336-6113
51 M 1902 77 FP

SHARON SPRINGS—913
(Northwest Kansas Society)

CHUNG, JOHN J, WALLACE CO MED CLINIC,67758
852-4214
23 M 58301 48 FP

SHAWNEE MISSION—913
(Johnson County Society)

ALLEN, MAX S, 5103 W 96TH TERR,66207
588-6988
11 M 1902 37 IM
ALTENBERND, ELVIN CONRAD, 7319 W 81ST,66204
648-2010
26 M 1902 54 FP
ALVAREZ, LUIS A, 10211 MISSION RD,66208
649-3400
32 M 64914 61 FP

ARMBRUSTER, ALBERT A, 9119 WEST 74TH SUITE 202,66204
362-9220
17 M 512 55 GS
ATHON, MERRILL D, 8501 OELMAR LANE,66207
642-4242
24 M 1902 54 FP
BADEEN II, LOUIS JOHN, 10550 QUIVIRA RD,66215
492-3344
49 M 2846 74 OPH
BAEKE, JOHN O, 6806 WEST 83RD,66204
642-4242
19 M 1902 52 FP
BAKER, WILLIAM STEVEN, 7700 W 63RD,66202
262-1843
47 M 702 73 P
BALANOFF, ARNOLD Z, 4601 W 109TH SUITE 122,66211
642-4040
42 M 1803 67 PD
BANSAL, ROOPA D, 8901 W 74TH SUITE 147,66204
384-2220
37 F 49504 64 FP
BANSAL, SATISH C, 8901 W 74TH SUITE 147,66204
384-2220
38 M 49541 61 ORS
BAPTIST, JEREMY E, 5811 OUTLOOK,66202
432-0625
40 M 2846 78 A
BARE II, CHARLES E, 8901 W 74TH SUITE 353,66204
677-2460
43 M 1902 69 U
BARKER, ELIZABETH B, 4121 WEST 83RD SUITE 123,66208
381-6669
30 F 4706 55 P
BARKER, JAMES BERTON, 8901 W 74TH,66204
362-6310
31 M 4706 55 OTO
BARNETT JR, THOMAS E, 10550 QUIVIRA SUITE 290,66215
492-2555
52 M 1902 75 IM
BARNHART, RONALD J, 9119 WEST 74TH,66204
831-2334
41 M 2501 68 OBG
BARNHORST, DONALD A, 10550 QUIVIRA STE 510,66215
492-6200
37 M 2834 63 CDS
BARR, RICHARD N, 7301 MISSION ROAD,66208
432-4366
32 M 1902 57 OPH
BARRICK, BRUCE, SH MISSION MEDICAL CENTER,66201
676-2340
39 M 1902 65 PATH
BATTY, LARRY H, 9119 W 74TH,66204
831-2334
51 M 1902 77 OBG
BATTY, THOMAS V, 5555 W 58TH,66202
432-2080
21 M 3806 54 FP
BAUER, LAFE W, 4818 W 80TH,66208
642-5369
20 M 1902 49 ANES
BAUER, MARTIN L, 2500 W 71ST,66208
281-8881
47 M 1902 73 PD
BAXTER, KIRKMAN G, 5013 WOODSON,66202
-
57 M 1902
BEAMON, RICHARD F, SUBURBAN MEDICAL CENTER,66215
492-1000
47 M 2803 73 EM
BELL, DELORIS W, 4601 W 109TH SUITE 116,66211
341-6550
42 F 1902 68 OPH
BELZER, EDWARD G, PO BOX 7426,66207
381-8282
36 M 3005 58 PD
BERRY, JOHN M, 8800 W 75TH SUITE 320,66204
262-3288
47 M 1902 74 D
BETTEGA, PAULO R, 8500 W 110TH,66210
648-5303
47 M 18708 70 P
BIKALES, VICTOR WILLIAM, 7301 MISSION RD,66208
384-1311
13 M 2105 38 P
BISHOP, FRANCIS E, 7501 MISSION ROAD,66208
648-3533
20 M 1902 45 P
BLETZ, DONALD B, 10550 QUIVIRA SUITE 510,66215
492-6200
28 M 5104 58 IM
BOLES, J MICHAEL, 5949 NIEMAN ROAD,66203
631-1300
35 M 1902 61 FP

BRAVERMAN, DAVID ELLIOTT, 4601 W 109,66211
341-1101
47 M 2507 72 PDO
BRIDGENS, JAMES G, SH MISSION MEDICAL CENTER,66201
676-2340
22 M 1902 47 PATH
BROUCEK, FRANCIS J, 4121 W 83RD,66208
642-6845
32 M 1643 58 P
BROWN, WILLIAM R, 7301 MISSION RD,66208
236-8866
23 M 1902 48 IM
BROXTERMAN, STEVEN JOSEPH, 8901 W 74TH SUITE 225,66204
362-5510
51 M 1902 76 FP
BRUNING, ROGER MARION, 7301 MISSION SUITE 342,66208
384-0745
48 M 1902 70 FP
BUCKMAN, MARTIN SPALDING, 10550 QUIVIRA SUITE 290,66215
492-6200
49 M 2802 76 IM
BURGER, PAUL B, 5638 NIEMAN RD,66203
631-6114
25 M 2834 50 FP
CALKINS, JOHN W, 5213 ROSEWOOD DR,66205
-
51 M 1902 76 OBG
CALKINS, LARRY L, 5635 SUWANEE RD,66205
-
18 M 1902 43 DO
CARDUFF, JAY J, 6300 GLENWOOD,66202
432-4480
25 M 3006 54 FP
CARRASCO, LENOR C, 8971 W 75TH,66204
341-2000
41 F 74801 63 PD
CARTY, RUSSEL W, 7732 W 152ND CT,66223
-
53 M 1902
CASTEEL, CHARLES K, 8901 W 74TH SUITE 357,66204
831-1003
34 M 3901 59 U
CATTANEO, ERNEST A, 6100 MARTWAY,66202
262-3930
39 M 1902 65 IM
CAVITT, ROBERT F, 9119 WEST 74TH,66204
831-0700
24 M 1902 48 GS
CEDERLIND, CRANSTON JAY, 8901 W 74 SUITE 36,66204
236-6455
45 M 1902 71 OBG
CHANG, SHU FANG, 10200 W 75TH,66204
432-7885
25 F 24239 49 P
CHRISTIAN, STANLEY J, 9129 DELMAR,66207
-
18 M 1902 44 DO
CLENDENIN, ROBERT KEELE, 4801 W 110TH SUITE 200,66210
341-2100
48 M 1902 73 EM
CLYMER, DAVID J, 2309 W 71ST TERR,66208
-
53 M 4705 80 DRS
COE, RICHARD O, 7301 MISSION RD SUITE 247,66208
362-8505
31 M 4804 56 OPH
COHEN, MARC O, 8800 W 75TH SUITE 300,66204
362-1226
51 M 1001 77 IM
COHEN, ROBERT A, 3700 W 83RD,66208
642-2100
39 M 2803 64 PD
COHN, STEVEN G, 8627 LINDEN DR,66207
334-2500
41 M 1902 67 ANES
COLEMAN, ROBERT L, 8901 W 74TH SUITE 1,66204
362-0100
41 M 4113 66 PS
COOLEY, DAVID A, 8800 W 75TH SUITE 300,66204
362-1226
40 M 2802 66 RHU
COOPER, JACK R, 7301 MISSION RD SUITE 330,66208
432-6300
17 M 3840 43 NS
CORBIN, MURRAY D, 10550 QUIVIRA RD-5TH FL,66215
492-6200
39 M 1902 65 IM
COULTER, HENRY F, 4203 W 151 ST,66224
-
23 M 1902 51 DO
COULTER, THOMAS B, 8800 W 75TH SUITE 310,66204
677-3113
38 M 1205 64 OPH

COX JR, IRA, 5829 WOODSON PD BOX 975,66201
722-1100
19 M 1902 49 FP
CURRAN, KEVIN E, 4121 W 83RD,66208
649-9383
39 M 2803 65 OPH
DAVIS, RICHARD E, 8919 MISSION RD,66206
648-5303
26 M 1902 54 P
DELP, MAHLON H, 6131 TERRYDALE RD,66202
-
03 M 1902 34 IM
DENISON, TERRY R, 5811 OUTLOOK,66202
432-0625
29 M 1902 56 PD
DERRINGTON, KENNETH L, FOX HILL MED BLDG,66211
341-3535
44 M 1902 71 FP
DOCKHORN, ROBERT J, 5300 W 94TH TERR,66207
381-4674
34 M 1902 60 PDA
DOHERTY, WILLIAM R, 7600 STATE LINE,66208
649-3900
20 M 3006 56 FP
DUCKETT II, THOMAS G, 4601 W 109TH SUITE 116,66211
648-1022
41 M 1902 67 OPH
EIKERMANN, WILLIAM C, 9400 MISSION RD,66206
642-5184
42 M 1902 69 P
ENDERS, WRAY, 9034 COTTONWOOD DR,66215
-
02 M 1902 36 ANES
ESRIG, HAROLD L, D.D., 8132 SAGAMORE,66206
381-5033
30 M 2878 60 ANES
ETZENHOUSER III, RUSSELL O, P O BOX 7426,66207
381-8282
34 M 1902 59 PD
EVANS, CAROL ANN, 6100 MARTWAY SUITE 2,66202
362-0000
54 F 2846 78 IM
EVANS JR, WILLIAM E, 8741 HIGH DRIVE,66206
362-7363
24 M 1902 58 FP
FERGUSON, ROBERT LEON, 8901 W 74TH SUITE 208,66204
362-0300
47 M 1902 73 IM
FORDYCE, NORMAN, 8901 W 74TH,66204
722-0020
41 M 1902 67 DTD
FRANCISCO, CLARENCE L, 3509 W 85TH,66206
371-6802
09 M 1902 34 DRS
FULLEN, JERYL G, 8901 W 74TH SUITE 124,66204
831-2604
43 M 401 68 DRS
GARCIA, FRANCISCO, 8020 SANTA FE,66204
642-5000
32 M 27501 60 FP
GARDNER, GLENN M, 5200 WEST 64TH,66208
-
35 M 2803 60 IM
GENTRY, KALE C, 5105 W 84TH,66207
632-4242
31 M 1902 60 FP
GILBERT, ROBERTA M, GEORGETOWN MEDICAL BLDG,66204
341-1234
35 F 3506 62 P
GILLEN, BILLY A, 8802 BIRCH LANE,66207
-
29 M 1902 54 ANES
GOLDSTEIN, GERALD L, 4601 W 109TH SUITE 318,66211
383-3630
47 M 16504 76 P
GOLLERKERI, MOHAN P, 7301 MISSION RD STE 339,66208
236-8866
30 M 49516 52 HEM
GOMEZ, FRANCISCO, 4200 SOMERSET #160,66208
649-7300
15 M 26401 43 P
GOOD, WENDELL LISLE, 4601 W 109TH,66211
649-3883
24 M 1902 48 FP
GOSALIA, ANIL V, 11701 MACKEY,66210
334-2500
46 M 49501 64 GP
GRAHAM, SUSAN B, 6423 RILEY,66202
-
F 1902
GREGORY, RALPH W, 4601 W 109TH SUITE 222,66211
381-8823
24 M 1902 54 GS

GROSSMAN, HARVEY M, 4601 W 109TH SUITE 122,66211
642-4040
49 M 1902 74 PD
GRUNDMEIER, ANNETTE M, 10550 QUIVIRA SUITE 250,66215
492-2013
46 F 1611 77 PD
HACKER, DAVID CHARLES, 6900 W 67TH,66202
588-6670
50 M 1902 75 ANES
HAMTIL, LAWRENCE W, 10550 QUIVIRA RD,66215
649-6722
36 M 2803 61 PD
HANDLEY, DENNIS MICHAEL, 8650 W COLLEGE BLVD,66210
649-1311
50 M 2803 76 FP
HARMS, ALBERT C, 5750 WEST 95TH,66207
381-5550
13 M 1902 38 FP
HARPSTER, GENE O, 4121 W 83RD SUITE 216,66208
648-1400
31 M 1902 57 GS
HARTONG, WILLIAM A, 8901 W 74TH STE 372,66204
831-9300
44 M 1902 71 IM
HASTINGS, MARY T, 2520 W 50TH PL,66205
-
45 F 1902 77 D
HATHAWAY, PETER, 11055 CEDAR STE 216,66211
383-2270
31 M 3503 60 IM
HAY, JAMES R, 7720 W 85TH #101,66212
-
58 M 1902
HENRY, JOSEPH E, 8901 W 74TH #348,66204
432-8000
42 M 1902 68 PUD
HESSER, HERBERT H, 7207 EDGEWOOD BLVD,66203
-
06 M 1902 34 OO
HILL, RODNEY W, 8901 W 74TH SUITE 208,66204
362-6161
47 M 1902 74 IM
HITCHCOCK, C THOMAS, 8901 W 74TH SUITE 356,66204
677-2508
47 M GS
HOBSON, MILBURN W, 9119 W 74TH ST,66204
831-2334
30 M 1902 55 OBG
HODES, HERBERT C, 5007 W 112TH,66211
381-6868
43 M 1902 69 OBG
HODGES, BRUCE E, 10550 QUIVIRA SUITE 430,66215
888-0777
32 M 1902 63 FP
HOPKINS, WILLIAM O, 8800 W 75TH SUITE 350,66204
831-3500
33 M 2803 61 ORS
HORSEMAN, ROBERT F, 9119 W 74TH ST,66204
432-7419
19 M 1902 44 OBG
HOUSTON II, LAWRENCE MORLEY, 8650 W COLLEGE BLVD,66210
649-1311
50 M 2803 76 FP
HUANG, GEORGIANA L W, 6300 GLENWOOD #3,66202
384-4998
41 F 4107 75 GP
HUMPHREY, LOREN JENKINS, HUMPHREY MED CL & TUM INS,66204
384-2850
31 M 1611 56 SON
INNES, ROBERT C, 1022 BRIAR,66207
-
25 M 2802 49 OO
ITURRALDE, GEORGE, 7501 MISSION RD,66208
648-4949
21 M 13201 49 P
JACKSON, ROBERT V, 8901 W 74TH SUITE 150,66204
362-1660
49 M 2803 77 PD
JANES, DONALD R, 10550 QUIVIRA #360,66215
492-1955
34 M 1902 60 OBG
JONES, CHARLES E, SHAWNEE MISSION MED CNTR,66201
676-2214
31 M 1902 60 FP
JONES, H IVOR, 8901 W 74TH SUITE 269,66204
362-4040
24 M 80303 51 P
JOUVENAT, NEIL C, 10550 QUIVIRA SUITE 120,66215
492-1844
43 M 3005 71 OBG
KADIAN, RAJESH S, 10550 QUIVIRA SUITE 260,66215
541-0577
50 M 71 IM

KAGAN, STUART M, 10550 QUIVIRA - SUITE 340,66215
492-1111
44 M 4901 69 PD
KARLIN, RONALD J, 9336 W 49TH,66203
556-3000
54 M 1902 79 EM
KASHYAP, BANSHI PRASAD, 8901 W 74TH SUITE 257,66204
236-4500
47 M 49554 69 IM
KELLEY, MARSHALL D, 7232 GRANDVIEW DR,66204
-
59 M 1902
KENNEDY, KENNETH R, 6100 MARTWAY STE 11,66202
432-0126
24 M 1902 53 FP
KETCHUM, LYNN D, 10550 QUIVIRA RD STE 310,66215
492-3737
36 M 2101 60 PS
KHURANA, SATISH K, 9119 W 74TH SUITE G-2,66204
432-3334
41 M 49536 65 PD
KIMURA, CHARLES C, 8901 W 74TH SUITE 125,66204
262-4220
25 M 2101 56 A
KODANAZ, A AYTEKIN, 5710 REINHARDT DR,66205
334-2500
28 M 90201 55 ANES
KOZIKOWSKI, BEN M, 7301 MISSION RD,66208
362-8317
30 M 2834 55 ORS
KUBIN, DORIS A, 2504 W 71ST,66208
-
15 F 1902 43 OO
KURTH, PAUL H, 5555 W 58TH,66202
432-2080
53 M 2507 77 IM
KURTH, ROBERT H, 5555 W 58TH,66202
432-2080
28 M 3005 53 IM
LAPI, RUTH M, 2012 STRATFORD RD,66208
-
14 F 4107 37 OO
LASH, RAY E, 8901 W 74TH SUITE 21,66204
722-0080
50 M 1902 75 CD
LEATHERS III, HOLLIS K, SHAWNEE MISSION MED CNTR,66201
676-2340
38 M 3901 64 PATH
LEGASPI JR, PEDRO L, SHAWNEE MISSION MED CNTR,66201
676-2479
36 M 74801 60 ANES
LERNER, SCOTT A, 8901 W 74TH #348,66204
432-8000
47 M 1902 73 PM
LESTER, JOHN BUCKLES, 4140 W 71ST SUITE 108,66208
432-7276
45 M 1902 70 P
LEWIN, WALTER, 8901 W 74TH ST,66204
362-4040
30 M 1902 56 P
LEWIS, JAMES E, 3700 W 83RD SUITE 203,66208
649-0923
37 M 2101 63 P
LIPSEY, JAMES H, 8800 W 75TH SUITE 350,66204
831-3500
31 M 1606 56 ORS
LOHRENTZ, LOIS H, 2800 W 50TH TERR,66205
682-1189
26 F 1902 52 ANES
LULO, ANTONIO R, 7600 STATE LN,66208
649-3900
35 M 30801 60 IM
MACDOUGALL, MARGARET L, 6251 ASH,66202
588-6983
48 F 1901 77 NEP
MALLORY, JOHN A, 10550 QUIVIRA SUITE 510,66215
492-6200
43 M 2803 71 IM
MANLEY, JOSEPH W, 4601 W 109TH SUITE 306,66211
381-8838
42 M 1902 69 OBG
MANTZ, FRANK A, 9309 W 103RD,66212
-
12 M 4101 38 OO
MARR, GARRETT L, 7301 MISSION RD SUITE 225,66208
362-0582
44 M 1611 76 GS
MARVIN, NORMAN G, WYCLIFF SHOPPING CENTER,66212
541-8282
29 M 1902 56 FP

MASER, GEORGE R, 5808 NALL,66202
-
12 M 1902 36 DO
MATHEWS, ROBERT MAJOR, 7301 MISSION RD,66208
362-6888
25 M 1902 54 GS
MAXWELL, ROBERT A, 8901 W 74TH - SUITE 150,66204
362-1660
46 M 1902 73 PO
MCCAUGHEY, HUGH W, 11055 CEDAR SUITE 210,66211
381-1724
28 M 1902 53 IM
MCCOWEN, HERBERT M, 4835 W 62ND TERR,66202
-
58 M 1902
MCEACHEN, WILLIAM H, 3700 WEST 83RD SUITE 102,66208
649-3335
32 M 1902 59 PO
MCELROY, MICHAEL B, 6300 GLENWOOD,66202
432-1122
43 M 2803 69 DRS
MCGURK, THOMAS E, 4601 W 109 SUITE 206,66211
649-2080
39 M 2803 65 P
MCWHERTER, LOTTIE B, 5920 NALL SUITE 308,66202
362-1464
30 F 1902 57 IE
MENEZ, CESAR V, 4121 W 83RD SUITE 120,66208
381-4484
36 M 74810 56 P
MEYER, MARK E, 10308 W 62ND #6,66203
677-4818
58 M 1902
MILLER, FREEMAN LANCE, 10550 QUIVIRA SUITE 340,66215
492-1111
48 M 1902 74 PD
MISKEW, DON B W, 7301 MISSION RD SUITE 348,66208
362-8317
42 M 6506 69 DRS
MOFFAT, ROBERT E, PO BOX 4250,66204
677-0883
42 M 1902 68 OR
MORITZ, RICK S, 10806 WEST 98TH,66214
371-4343
54 M 1902 78 OR
MORONEY, JEAN M, 10550 QUIVIRA SUITE 510,66215
492-6200
25 F 4107 65 N
MUEHLBERGER, JAMES J, 4601 W 109TH SUITE 314,66211
383-3222
34 M 3006 60 PO
MUELLER, J KENT, 3700 WEST 83RD SUITE 203,66208
649-0923
35 M 1902 62 P
MURPHY, JAY W, 8901 W 74TH SUITE 208,66204
362-6161
49 M 3840 73 CO
MURRAY, W LEE, 4601 W 109TH SUITE 225,66211
381-2931
35 M 1902 61 DPH
NASH, ROBERT A, 4601 W 109TH STE 204,66211
649-8686
31 M 1902 55 P
NAUER, PAULA LOU, 7301 MISSION RD STE 342,66208
384-0745
49 F 1902 74 FP
NAVICKAS, LEONARD A, 8901 W 74TH SUITE 225,66204
362-5510
53 M 1902 77 FP
NEIBURGER, JAMES B, 11111 NALL AVE STE 114,66211
341-8907
46 M 1642 72 A
NEIGHBOR, ERNEST H, 7301 MISSION RD,66208
831-1100
40 M 1902 66 ORS
NELSON, BRYAN C, 9119 W 74TH,66204
384-5500
50 M 1902 75 PD
NELSON, JOHN B, 10550 QUIVIRA SUITE 510,66215
492-6200
48 M 2846 75 PM
NIEMAN, JOHN L, PO BOX 7426,66207
381-8282
28 M 3806 58 PO
NOSTI, JUAN C, 8901 W 74TH #345,66204
262-5014
38 M 13204 63 PS
NYE, C ERIK, 7301 MISSION RD SUITE 348,66208
362-8317
39 M 3520 65 DRS
O'BRYAN, JAMES J, 10550 QUIVIRA,66215
492-2525
47 M 1902 73 PD

O'CONNELL, FRANK A, 7830 STATE LINE,66208
381-0886
25 M 1902 51 FP
OKTAWIEC, DANUTA, 5848 FONTANA DRIVE,66205
-
22 F 80303 50 ANES
OWENS, RICHARD L, 10000 W 75TH SUITE 103,66204
362-3687
24 M 3006 51 OM
PATTERSON, JOHN R, 5317 CHADWICK RD,66205
-
20 M 1902 48 DO
PEARCE, EUGENE W J, 9119 W 74TH SUITE 104,66204
722-3102
24 M 2802 49 OBG
PEARCE, LUNETTA M, 9119 W 74TH SUITE 104,66204
362-1525
26 F 3005 49 FP
PENTECOST, RICHARD L, 8900 STATE LINE SUITE 350,66206
383-1410
32 M 1001 56 P
PETELIN, JOSEPH B, 8901 W 74TH SUITE 356,66204
677-2508
49 M 1902 76 GPVS
PETERSEN, A GENE, 3700 W 83RD,66208
648-3911
27 M 1902 54 IM
PETERSEN, GERALD D, 3700 WEST 83RD,66208
648-3911
30 M 1902 60 IM
PETIT, CARL ALFONSO, 10550 QUIVIRA SUITE 240,66215
888-1114
33 M 60 GS
PETRIE, SAMUEL C, GEORGETOWN MED BLDG,66202
722-1166
27 M 1902 58 IM
PFUETZE, BRUCE L, 4601 W 109TH,66211
383-3630
42 M 1902 68 A
PHILLIPS, WARREN G, 3700 W 83RD,66208
649-0923
26 M 1902 60 P
PILCHARD, WILLIAM A, GEORGETOWN MED BLDG,66204
362-3210
39 M 1602 65 OPH
PINGLETON, WILLIAM WARREN, 8901 W 74TH #348,66204
432-8000
42 M 3901 67 PUO
PITTS, RONALD L, 8901 W 74TH - SUITE 330,66204
362-2524
35 M 2002 62 D
PODNAWALA, HUSENI E, 3001 W 121,66209
561-2025
33 M 49528 59 P
POWELL, CAROL W, 8216 CHEROKEE CIRCLE,66206
381-3785
25 F 1902 51 P
POWELL, KENNETH A, 8216 CHEROKEE CIRCLE,66206
753-7000
25 M 1902 53 IM
PRONKO, MICHAEL J, 4121 W 83RD SUITE 223,66208
648-7878
34 M 1902 60 P
RALSTIN, JAMES H, 14708 W 71ST TERR,66216
299-2069
49 M 1902 74 IM
REIVICH, RONALD S, 8900 STATE LN #331,66206
383-3050
34 M 3806 60 P
RICE, BERNARD F, 8901 W 74TH SUITE 125,66204
262-9222
31 M 4113 56 END
RICHTER, DON G, SHAWNEE MISSION MED CTR,66201
676-2464
50 M 1902 79 ANES
RICK JR, GREGORY G, THE GEORGETOWN MED BLDG,66204
831-9300
40 M 1902 66 GE
RIFFEL, LAWRENCE D, 10550 QUIVIRA SUITE 510,66215
492-6200
53 M 1902 78 IM
RITCHIE, KAREN S, 8901 W 74TH SUITE 21,66204
722-0080
49 F 3840 73 P
ROBERTSON, EDWARD J, SHAWNEE MISSION MED CTR,66201
676-2479
46 M 1902 78 ANES
ROBINSON, JOHN D, SHAWNEE MISSION MED CTR,66204
676-2000
48 M 1902 74 ANES
ROBISON, JAMES T, 8800 W 75TH SUITE 310,66204
677-3113
21 M 4812 45 OPH

ROMITO, CYNTHIA L, 5811 OUTLOOK,66202
432-0625
47 F 1902 72 A
ROSENBERG, STANTON L, 1900 W 75TH SUITE 200,66208
362-8080
30 M 1902 55 R
ROSENTHAL, RICHARD, 5102 W 112TH ST,66211
492-1000
50 M 2820 76 EM
RUBIN, HERBERT M, 10550 QUIVIRA - SUITE 340,66215
492-1111
37 M 2803 63 PO
RUMOLD, MERVIN J, 6340 INDIAN LANE,66208
-
03 M 1902 30 GS
RYAN, MICHAEL E, 4601 W 109TH SUITE 210,66211
341-4030
46 M 1902 72 N
RYMER, ROBERT A, 8901 W 74TH SUITE 373,66208
722-0170
41 M 0702 68 ORH
SACHEN, FREDERICK L, 9620 ROE,66207
-
45 M 4813 79 N
SAFFO, KARL S, 8901 W 74TH,66204
362-9585
39 M 52801 62 RS
SATHYANARAYANA, SARASWATHI, 8901 W 74TH SUITE 20,66204
677-2281
45 F 49509 67 OBG
SAWKAR, LAXMIDAS A, 8901 W 74TH SUITE 312,66204
384-4844
36 M 49523 63 ONC
SCHAEFER, JOSEPH PETER, 10550 QUIVIRA - SUITE 230,66215
492-7440
34 M 1902 60 IM
SCHROLL, JOHN T, 8901 W 74TH SUITE 248,66204
384-4990
51 M 1902 76 OBG
SCLAR, WILLIAM C, DOCTORS BLDG SUITE 450,66215
492-7730
46 M 2501 72 GS
SHAAD, DOROTHY J, 2322 W 51ST,66205
-
09 F 1902 44 OD
SHAFFER, KATHLEEN BRAY, 9119 W 74TH SUITE 107,66204
384-5500
54 F 2846 79 RO
SHAFFER, STANLEY G, 9119 W 74TH SUITE 107,66204
384-5500
55 M 2846 79 PO
SHERIDAN, RANDY M, 8901 W 74TH SUITE 36,66204
236-6455
53 M 1902 78 OBG
SHOFSTALL, WILLIAM H, 6100 MARTWAY,66202
-
11 M 3901 41 OD
SILLS, THOMAS D, 14809 W 85TH TERR,66215
281-8883
49 M 5606 77 EM
SILVER, BRADD J, 8901 W 74TH SUITE 30,66204
432-2280
50 M 1205 76 IM
SMITH, DALE C, 4601 W 109TH ST SUITE 224,66211
381-0353
20 M 1902 45 OPH
SMITH, DONALD J, 8600 W 95TH ST,66212
642-4515
18 M 1902 49 FP
SNODELL, FIRMIN E, 5555 W 58TH,66202
432-2080
31 M 1902 61 IM
SNOW JR, ARTHUR D, 8901 W 74TH - SUITE 225,66204
362-5510
45 M 1902 75 FR
SOELDNER, JAMES OLIVER, 8901 W 74TH SUITE 24,66204
722-6214
44 M 1902 70 FR
SPEER, FREDERIC, 5811 OUTLOOK,66202
-
09 M 1902 34 OD
STEINER, ROBERT M, 5311 W 96TH ST TERR,66207
677-0883
39 M 4112 65 R
STEINZEIG, ALFRED S, 6500 HODGES DR,66208
-
13 M 1902 37 OD
STEVENSON, E KENT, 4121 W 83RD SUITE 150,66208
649-5566
45 M 2802 67 CHP
STITT, RONALD W, 4221 W 104TH TERR,66207
342-7233
21 M 1902 45 U

STRIEBINGER, CHARLES M, 4601 W 109TH SUITE 307,66211
341-7299
45 M 1606 67 NS
STUBER, JACK LAWRENCE, RD BOX 4250,66204
677-0883
40 M 1902 66 OR
STUCKEY, CHARLES E, 10550 QUIVIRA SUITE 240,66214
492-7737
41 M 3005 68 GS
SUGAR, ROBERT L, 8901 W 74TH SUITE 248,66204
384-4990
40 M 3508 66 OBG
SULLIVAN, TOM G, 10550 QUIVIRA SUITE 320,66215
492-3600
44 M 1902 71 OBG
SULLIVAN JR, HENRY B, 5817 NIEMAN RD,66203
631-6160
24 M 1902 52 FP
SUTTON JR, RICHARD L, 3203 W 83RD TERR,66206
-
08 M 2501 29 OD
SZYMANSKI, DENNIS C, 10300 CATALINA,66207
561-4655
43 M 2507 70 NS
TALLEY, ROBERT L, 6501 W 101 ST PLACE,66212
588-3000
48 M 4802 74 IM
TENNY, ROBERT T, 4601 W 109TH SUITE 307,66211
341-7299
51 M 1902 76 NS
TETZLAFF, ARCHIBALD D A, 4520 W 65TH,66208
334-2500
26 M 40721 52 ANES
THOMSEN, GARY, 8901 W 74TH SUITE 225,66204
362-5510
51 M 3005 76 FR
TOALSON, WILLIAM B, 8901 W 74TH SUITE 208,66204
371-3515
37 M 1902 63 CD
TRETBAR, LAWRENCE L, 8901 W 74TH,66204
677-1776
33 M 1902 60 GS
TROPP OD, ARNOLD L, 8600 W 95TH STE 102,66212
648-5600
46 M 2878 72 FP
TUCKER, SHERIDAN G, 4121 W 83RD SUITE 150,66208
649-5566
50 M 1902 75 R
TUTERA, GINO, 10550 QUIVIRA SUITE 120,66215
492-1844
45 M 2803 71 OBG
VALK, WILLIAM L, 5401 W 81ST,66208
588-6146
09 M 2501 37 U
VANNAMAN, DONALD D, P O BOX 7426,66207
381-8282
43 M 1902 71 RD
VENNEMAN II, CHARLES R, 10505 W 89TH,66214
-
53 M 1902
WALL PHD, LEONARD L, 5011 NEOSHO AVE,66205
384-3342
50 M 1902
WANG, SIDNEY W, 10550 QUIVIRA SUITE 130,66215
492-1500
32 M 38503 58 FP
WEBB, JAMES R, 5949 NIEMAN ROAD,66203
631-0900
34 M 1902 61 FP
WERTH, CLAUDE J, 4121 W 83RD,66208
649-5566
37 M 1902 64 P
WHITEHEAD, RICHARD E, 7301 MISSION RD,66208
362-8317
31 M 2501 58 ORS
WHITLEY, DOUGLAS M, 4601 W 109TH SUITE 202,66211
341-4770
34 M 1902 60 D
WIGGINTON, GERALD D, D.O., 9119 W 74TH,66204
384-5500
44 M 2878 70 RD
WILEY, JOHN H, 9119 W 74TH SUITE 201,66204
831-2334
37 M 4113 63 OBG
WILSON, ROBERT B, 6915 W 51ST PLACE,66202
-
10 M 1902 40 OD
WILSON, SLOAN J, 5618 W 62ND,66202
-
10 M 1902 36 HFM
WOODS, DENNIS O, 5435 BRIAR,66205
-
60 M 1902

WURSTER, GEORGE R, 3700 W 83RD SUITE 203,66208
649-0923
35 M 1902 61 P
YE, RICHARD C, 7301 MISSION RD,66208
362-7505
20 M 24222 46 PS
YOHE, RUTH M, 8600 W 95TH ST,66212
383-3377
26 F 4107 54 POA
YOUNG, JOHN W, 8220 TRAVIS STE 115,66204
383-1550
37 M 4706 63 PS
YOUNGSTROM, KARL A, 9660 REEDER PLACE,66214
-
08 M 3607 44 OO
ZACK, ASHLEY S, 4601 WEST 109TH SUITE 122,66211
642-4040
46 M 2803 73 PO
ZAMIEROWSKI, DAVID S, 8800 W 75TH SUITE 340,66204
831-4113
42 M 2307 68 PS
ZIMMERMAN, DANIEL O, 4761 RAINBOW,66205
831-0910
45 M 3005 70 IM

SMITH CENTER—913
(Central Kansas Society)

SHEPPARD, ROBERT G, 120 E COURT,66967
282-6654
21 M 1902 45 GS
STEINKRUGER, VERLYN WILLIAM, 120 E COURT,66967
282-6654
28 M 3005 53 FP
WOODS, HUGH J, 120 EAST COURT,66967
282-6654
26 M 1902 52 FP

SOUTH HAVEN—316
(Tri-County Society)

UBELAKER, ERNEST J, ,67140
892-2261
11 M 1902 38 FP

SPEARVILLE—316
(Barton County Society)

QUACKENBUSH, ROBERT P, SPEARVILLE DIST HOSP,67876
549-3471
21 M 1611 52 FP

ST. FRANCIS—913
(Northwest Kansas Society)

CRAM, ERNEST R, PO BOX 625,67756
332-2126
24 M 1902 52 FP
JEWELL, ROSS L, 203 SPENCER,67756
332-2832
13 M 1902 56 FP
STEPHENSON, LUCILLE C, ,67756
-
06 F 1902 32 FP
WALZ, THOMAS J, 115 SOUTH QUINCY,67756
-
94 M 1902 21 OO

ST. MARYS—913
(Pottawatomie County Society)

BROWN, FRED E, 602 W PALMER,66536
437-2256
26 M 1902 55 FP

STAFFORD — 316
(Ninnescah Society)

BROWN, C EVERETT, 102 N MAIN,67578
234-5251
10 M 1902 47 A
QUIJANO JR, RAMON S, 412 E GRAN,67578
234-5236
45 M 74811 71 GP

STERLING—316
(Rice County Society)

OYSART, JACK C, 224 N FOURTH,67579
278-2181
12 M 3901 39 OO
SIMPSON, TOM C, 239 N BROADWAY,67579
278-2123
47 M 1902 73 FP

STOCKTON—913
(Central Kansas Society)

MAUCK, HAROLD C, 623 SOUTH 2ND,67669
425-6280
20 M 1902 54 FP
VOTAPKA, WILLIAM L, 623 S SECOND,67669
425-6280
24 M 1902 53 FP

SUBLETTE—316
(Southwest Kansas Society)

THIEMANN, A H, D.O., 301 DERBY,67877
675-2241
12 M 1805 42 FP

SYRACUSE—316
(Southwest Kansas Society)

FELDMEYER, SEELEY T, PO BOX 1087,67878
384-7735
46 M 74811 80 GP
PETTERSON, CECIL E, PROFESSIONAL ASSOCIATION,67878
384-5731
14 M 1902 39 FP

TONGANOXIE—913
(Leavenworth County Society)

STEVENS, PHILIP L, BOX 319,66086
845-2090
27 M 1902 54 FP

TOPEKA—913
(Shawnee County Society)

AGAN, LAWRENCE M, 1314 PEMBROKE LN,66604
357-4306
20 M 5002 44 R
ARREDONDO, MARIO, 6024 SW 26TH,66614
296-7216
25 M 26401 54 P
ARTZER, DENNIS C, 901 GARFIELD,66606
354-9591
51 M 1902 76 IM
ARUNAKUL, PUNYA, 1710 W TENTH,66604
234-2624
44 M 89104 69 OTO
ASHLEY, BYRON J, 3222 PLASS,66611
233-2280
98 M 1902 24 OPH
ASHLEY JR, B JOHN, 1616 WEST 8TH ST,66606
233-2280
31 M 1902 56 OPH
ATLURU, NARAYANA RAO, 5207 W 11TH TERR,66604
232-8761
47 M 49550 69 ANES
AVERILL, STUART C, MENNINGER FD,66601
273-7500
24 M 502 52 P
BAEHR, RALPH H, S-F CAP REG RADIOTHER CTR,66606
234-3451
35 M 1606 59 R
BAIR, GLENN O, 634 MULVANE SUITE 400,66606
233-5153
31 M 2401 57 IM

BAKER, FREDERICK C, 2101 WEST 10TH,66604
232-0909
35 M 4113 62 FP
BAKER, PHILLIP L, 300 CONT MED BLDG,66606
357-0301
37 M 3005 63 ORS
BAKER, RAY D, 1615 W EIGHTH,66601
233-8961
30 M 4812 55 GPM
BARABAN, MARC R, 1319 HUNTOON,66604
357-5325
50 M 2846 75 PS
BAUCOM, KARAN YVONNE, 1516 W SIXTH,66606
233-1639
50 F 1902 75 OBG
BAUCOM-COPELAND, SHARON LAVARNE, 634 MULVANE STE 402,6660
235-9545
50 F 2846 75 FP
BAUDE, EUGENE L ANDRE, 1815 WESTWOOD CIRCLE,66604
354-1946
02 M 39606 27 IM
BEACH, RICHARD R, 106 MED ARTS BLDG WEST,66604
233-7943
23 M 2802 48 IM
BEALE, DAVID A, MENNINGER FD,66601
273-7500
31 M 5404 56 P
BECK, JOSEPH D, 2760 SW BURLINGAME,66611
-
18 M 3005 43 DD
BEDFORD, D R, PO BOX 1772,66615
-
09 M 4802 40 IM
BEELMAN, FLOYD C, 1286 LAKESIDE DR,66604
-
02 M 3840 35 FP
BELLER, WILLIS L, 3651 HOLLY LANE,66604
-
14 M 1902 41 OO
BERLAND, DAVID I, BOX 829,66601
273-7500
47 M 2803 78 P
BICKELHAUPT, ETHAN E, PO BOX 2033,66601
273-7500
52 M 5404 78 P
BLAKE, HENRY S, 1933 WESTWOOD DR,66604
-
11 M 3520 37 OO
BONEBRAKE, C RICHARD, 634 MULVANE STE 104,66606
233-1979
48 M 1606 75 OBG
BOREL, DAVID, ST FRANCIS HOSP & MED CTR,66606
295-8473
45 M 1902 71 PATH
BOWEN, CLOVIS W, 2200 WEST 10TH,66604
234-8601
12 M 1902 37 FP
BOWEN JR, HARRY J, 2200 WEST 10TH,66604
234-8601
11 M 1902 37 FP
BOYD, SPENCER H, 1815 WEST 2ND,66606
-
11 M 1902 35 OBG
BRAHMAN, HERBERT D, 1700 SEVENTH,66606
295-8471
43 M 512 70 PATH
BRAUN, ROBERT W, 901 GARFIELD,66606
354-9591
44 M 2803 70 IM
BRAUNSDORF, ROBERT L, 2843 VIRGINIA,66605
-
08 M 2501 35 FP
BRIDWELL, RUSSELL E, 1710 W TENTH,66604
234-2624
26 M 1902 51 ENT
BURDZIK, EBERHARD G, 2700 W SIXTH,66606
296-4222
26 M 40916 54 P
CACHIA, RICHARD M, DEPT OF PATHOLOGY,66606
295-8472
51 M 62701 73 PATH
CASHMAN JR, MAURICE R, 901 GARFIELD,66606
354-9591
35 M 1902 61 HEM
CAVANAUGH, JOHN W, 200 PROFESSIONAL BLDG,66604
235-3488
13 M 1803 39 GS
CHAMBERLIN JR, CECIL R, BOX 829,66601
273-7500
30 M 3901 55 CHP
CHEN, CHU-CHI, 918 W TENTH,66604
354-4465
47 M 24405 73 U
CHEN, TAK-MING, 303 MED ARTS BLDG,66604
234-3451
41 M 24402 68 ANES
CHERRY JR, ARTHUR C, 918 WEST 10TH,66604
233-3362
27 M 3806 53 PD
CLARK, CRAIG N, 3124 E 6TH,66607
354-7683
29 M 1902 58 FP
COCHRAN, PAUL W, MENNINGER FD,66601
273-7500
33 M 4802 58 IM
COHEN, LOUIS, 918 W 10TH,66604
233-7175
14 M 1902 41 IM
COKELEY, JOHN M, 2200 GAGE BOULEVARD,66622
272-3111
30 M 5104 55 P
COKER, W LAURENCE, 631 HORNE #340,66606
232-9394
53 M 1902 78 FP
COLLINS, DEAN T, MENNINGER FD,66601
273-7500
28 M 1902 55 P
COLLINS, EDWARD JOSEPH, 900 WASHBURN,66606
233-3242
45 M 1611 71 DPH
COLLINS, ELISABETH B, 634 MULVANE #402,66606
233-6399
21 F 40905 46 P
COLLINS, FRANCIS T, 206 MED ARTS BLDG EAST,66604
233-6470
14 M 1902 43 IM
COOLEY, DENNIS M, TOPEKA MEDICAL CENTER,66604
233-3362
51 M 1902 77 PD
COTTON, ROBERT T, 901 GARFIELD,66606
354-9591
19 M 1902 45 IM
CRARY, JOHN E, 201 MED PLAZA BLDG,66604
233-4202
18 M 1902 43 IM
CROUCH, STEVEN W, 904 MULVANE,66606
232-8224
51 M 1902 76 PD
CROUCH, WILLIAM H, 904 MULVANE,66606
232-8224
20 M 2802 45 PD
DAVIS, CHESTER R, 631 HORNE,66606
232-9394
50 M 1902 75 FP
DELCORE, ROMANO, BOX 829,66601
273-7500
30 M 64901 50 P
DELGADO, SERGIO, 634 MULVANE STE 200,66606
357-0352
37 M 2501 62 ORS
DESDIGNIE, RAFAEL R, 3107 W 21ST,66604
296-5306
19 M 27501 P
DUNAGIN, JACK A, 925 WESTERN,66606
233-7138
20 M 1902 44 P
DUNIVEN, PHILIP L, RADIOLOGY & NUCLEAR MED,66604
234-3451
52 M 4812 77 R
DURST JR, ROBERT D, CONTINENTAL MED BLDG,66606
357-5166
42 M 2803 69 D
EATON, EDWARD L, 925 WESTERN,66606
233-7138
40 M 0401 73 P
ELDER, DOUGLAS M, 310 MED ARTS BLDG,66604
234-3451
41 M 1902 69 DR
FAIRCHILD, RICHARD S, 901 GARFIELD,66606
354-9591
48 M 1902 76 IM
FEAGAN, JERRY, 901 GARFIELD,66606
354-9591
39 M 1902 63 GE
FERNANDEZ, LUIS A, PO BOX 118,66601
233-8961
14 M 27501 41 PD
FIELD, RICHARD A, 303 MEDICAL ARTS BLDG,66604
235-3451
29 M 1902 55 ANES

FILLMAN, ELOON M, 301 MEDICAL PLAZA BLDG,66604
233-4256
20 M 1611 44 U
FORD, FRED L, 220 MEDICAL ARTS BLDG,66604
234-5516
11 M 2501 36 GS
FOSTER, D BERNARD, 3111 JEWELL,66611
-
14 M 2501 38 NP
FUSILLO, MICHAEL, BOX 829,66601
273-7500
48 M 3508 74 P
GANDHI, SHANTIKUMAR K, 40 MED ARTS BLDG,66604
233-1710
40 M 49501 67 TS
GANZARAIN, RAMON C, MENNINGER FD,66601
273-7500
23 M 23101 47 P
GAY, JOHN D, 310 MED ARTS BLDG,66604
234-3451
42 M 4802 68 OR
GENDEL, JOSEPH E, 918 W 10TH,66604
235-9914
12 M 4804 37 ORS
GIESSEL, MICHAEL D, CONTINENTAL MED BLDG,66606
357-5166
48 M 1902 74 O
GIMPLE, KENNETH, 631 HORNE #200,66606
233-7491
45 M 1902 71 ORS
GLEASON, JIMMIE A, 800 LINCOLN,66606
233-5101
33 M 1902 58 OBG
GODFREY, KENNETH E, 9550 SW 53RD,66604
273-6153
18 M 3901 50 P
GOOTEE, JOSEPH E, 3204 W 17TH,66604
-
21 M 2834 47 OD
GRAHAM JR, CHARLES P, 631 HORNE #400,66606
354-9504
40 M 3601 65 GS
GRAY, DAVID E, PD BOX C-50,66601
234-8621
16 M 1606 42 GEN
GRAYBB, ANTOINE S, MEMORIAL HOSP,66607
354-5100
18 M 60501 46 IM
GREENBERG, MARK, 310 MED ARTS BLDG,66604
234-3451
46 M 1611 72 R
GREENE, HORACE T, 1710 W 10TH,66604
354-7508
15 M 401 42 FP
GREENWOOD, EDWARD D, BOX 829,66601
-
01 M 702 38 CHP
GREER, RICHARD H, 918 W 10TH,66604
233-5084
09 M 1902 39 OD
GUTOVITZ, ALLEN LOUIS, BROCK MEDICAL PLAZA,66606
233-9643
46 M 1611 72 CD
HACKER, ELAINE MARY, 3026 QUAIL CREEK,66614
296-3981
25 F 2604 50 OBG
HAINES, ROBERT A, 2700 SIXTH,66606
296-3436
16 M 3841 42 P
HALLEY, M MARTIN, 40 MED ARTS BLDG,66604
233-1710
27 M 2401 53 TS
HARRIS, HUBERT L, 210 MED ARTS BLDG,66604
233-3151
12 M 1803 39 O
HARRIS, PATRICIA A, 1617 W 26TH,66611
354-1906
29 F 1902 54 IM
HARRISON, HALL E, 901 GARFIELD,66606
354-9591
39 M 2802 65 IM
HARVEY, R CLAY, 1001 HORNE #310,66604
234-3451
52 M 1902 78 R
HEBBAR, SATYA N, BROCK MEDICAL PLAZA,66606
233-9643
39 M 49509 62 CD
HERRERA, JORGE J, 2825 CALIFORNIA,66605
267-5370
27 M 64901 55 IM
HICKMAN, JAMES STEPHEN, 634 MULVANE SUITE 103,66606
233-8508
53 M 2803 79 PD

HIEBERT, JOHN B, 901 GARFIELD,66606
354-9591
40 M 1902 68 CD
HILL, ROBERT N, 901 GARFIELD,66606
354-9591
14 M 1902 67 IM
HILST, WILBUR D, 1271 WOODHULL,66604
273-5870
31 M 3005 55 GS
HIRSCHBERG, J COTTER, MENNINGER FD,66601
273-7500
15 M 1602 40 CHP
HISZCZYNSKYJ, ROMAN, 1500 W TENTH,66604
354-6870
35 M 1803 66 PATH
HOBBS, DONALD D, 200 CONTINENTAL MED BLDG,66606
233-7491
28 M 2401 54 ORS
HOHERZ, DAVID G, 631 HORNE SUITE 220,66606
235-1170
45 M 1803 72 TS
HOLMES, ROBERT W, 901 GARFIELD,66606
354-9591
52 M 1902 74 IM
HORNE, JAMES B, 1305 LAKESIDE DR,66604
233-1730
26 M 1606 52 P
HOSTETTER, JAMES P, 1500 W TENTH,66606
354-6100
43 M 1902 69 EM
HOYT, ARTHUR W, 2055 CLAY,66604
234-5663
14 M 2501 40 P
HSU, CHENG H, 1516 W SIXTH,66606
232-1005
41 M 38502 66 U
HSU, SHIN-FU, MEDICAL PLAZA BLDG,66604
232-0362
43 M 24402 68 OTO
HUANG, JONSON, 901 GARFIELD,66606
357-6171
52 M 2701 77 N
HUSTON, JOSEPH W, 634 MULVANE,66606
357-0352
35 M 1902 62 ORS
HUTTON, FREDERICK A, 102 MED PLAZA BLDG,66604
234-0553
29 M 6701 58 PS
HYLAND, JOSEPH M, BOX 829,66601
273-7500
45 M 53902 68 P
ILIFF, R DOUGLAS, 1500 SW OAKLEY,66604
354-6100
49 M 1902 74 FP
ILORETA, ALFREDO T, 1516 W SIXTH,66606
232-1005
47 M 74801 71 U
ISAACSON, RICHARD N, 1001 GARFIELD STE 301,66604
233-4256
48 M 2501 75 U
JACKSON, LINDA H, 212 WOODLAWN AVE,66606
273-7500
42 F 3601 67 CHP
JACOBY II, ROBERT E, 340 CONT MED BLDG,66606
232-9394
46 M 2307 72 FP
JANSSEN, ERWIN T, MENNINGER FD,66601
273-7500
36 M 1803 62 P
JOHNSON, LEE ROY W, 2400 W 29TH,66614
267-0155
37 M 1606 63 P
JOSEPH, BRIAN W, 2115 W TENTH,66604
233-3326
38 M 35205 61 CHP
JOSS, CHARLES S, 221 MED ARTS BLDG W,66604
232-0444
14 M 1606 40 GS
JOYCE, G BERNARD, 4929 WEST HILLS DR,66606
-
17 M 1902 44 OD
KAVEL, KARL K, MED PLAZA BLDG,66604
234-2663
36 M 3605 64 PDA
KEARNS, NORBERT W, BOX 829,66601
273-7500
43 M 1002 70 P
KELLY, DAN A, 904 MULVANE,66606
232-8224
39 M 2803 64 PD
KEYS JR, ROBERT C, 303 MED ARTS BLDG,66604
235-3451
36 M 1902 62 ANES

KEYS SR, ROBERT C, 3034 LYOIA,66614
-
97 M 1902 27 OO
KIM, YONG W, CONT MED BLDG,66606
232-6964
28 M 58302 49 IM
KINOLING, PAUL H, 40 MED ARTS BLDG,66604
233-1710
30 M 3545 61 TS
KIRKEGAARD, RDDGER S, 835 WESTERN,66606
233-6493
30 M 1803 56 OPH
KLEINHOLZ JR, EMIL JOHN, 600 MAOISON,66607
354-5220
39 M 3503 65 IM
KLEMMER, HERBERT, MENNINGER FO,66601
273-7500
11 M 4102 37 P
KOONTZ, JUDITH A, BOX 829,66601
233-5033
49 F 1902 75 CHP
KOVARIK, ERNEST O, 900 WASHBURN,66606
233-1800
36 M 3005 64 OPH
KROLL, HARRY G, 200 CONTINENTAL MED BLDG,66606
233-7491
24 M 1602 50 DRS
LACCHEO, MICHAEL L, 2115 W 10TH,66604
354-4600
51 M 3840 76 FP
LAI, MAX G, 918 W TENTH,66604
354-4465
45 M 24405 72 U
LAI-CHEN, SHEUE-HUEY, 918 W TENTH,66604
354-4465
48 F 38505 73 GP
LAUNEY, WALTON S, 1001 HDRNE #310,66604
234-3451
39 M 4804 75 R
LAWSON, DWIGHT, 1203 SW 29TH,66611
-
06 M 2802 30 IM
LEE, CATHERINE, 600 MAOISON,66607
354-5224
49 F 2307 75 IM
LEE, SDNG DOW, 303 MED ARTS BLDG,66604
235-3451
43 M 38505 68 ANES
LEE, SONG PING, 918 W 10TH,66604
233-6001
34 M 38502 61 OTD
LEFEVRE, JOHN R, D.O., 3716 W 29TH #713,66614
-
51 M 2878 78 OS
LEGLER, GARY LEE, O.D., 2835 SE SKYVIEW CT,66605
233-8961
50 M 2878 77 GP
LEIFER, WILLIAM N, 1500 W TENTH,66606
354-6031
47 M 1902 78 PATH
LENTZ, WILLIAM R, 300 MEDICAL ARTS BLDG,66604
235-3443
24 M 1902 53 FP
LESSENDEN JR, C M, 5635 NW BRICKYARD RD,66618
272-3111
18 M 1902 43 D
LEVY, EDWIN Z, 4125 SW GAGE CTR OR L-#6,66604
273-5610
29 M 1606 54 P
LIESMANN, GEORGE E, 631 HDRNE,66606
235-1170
49 M 1902 74 GPVS
LIESMANN, JEAN ELIZABETH, MEMORIAL HOSP,66607
354-5275
49 F 1902 74 IM
LONG, JDHN W, 1001 HDRNE ST,66604
233-1710
43 M 1902 70 TS
LUMB, RAYMDND C, 901 GARFIELD,66606
354-9591
42 M 1001 68 RHU
LYNCH, JOHN A, 300 CDNT MED BLDG,66606
357-0301
30 M 2834 55 DRS
MARSHALL, B M, 1826 SW 34TH,66611
-
08 M 2802 34 U
MARTIN, WILLIAM O, 303 MED ARTS BLDG,66604
235-3451
19 M 1902 44 ANES
MAU, WALTER, 301 MEDICAL PLAZA BLDG,66604
233-4256
16 M 1611 40 U

MCCARTER, DUANE K, 2101 W 10TH,66604
233-8979
26 M 1902 58 IM
MCCLELLAN, JOHN W, 2701 BOSWELL,66611
-
11 M 3006 36 DO
MCCLURE, JAMES A, 1541 WESTOVER RO,66604
-
18 M 1902 44 OO
MCCOMAS JR, MARMADUKE O, 3020 BRUSH CREEK CIRCLE,66614
-
16 M 1902 43 OO
MCCOY, MICHAEL T, 631 HORNE SUITE 300,66606
357-0301
49 M 1902 75 ORS
MCELROY, ROBERT T, 221 MED ARTS BLDG,66604
232-0444
35 M 1902 61 GS
MCELROY, WILBUR J, 1616 W EIGHTH,66606
233-2280
35 M 1902 61 DPH
MECH, ARNOLD W, 3240 TIMBERLAKE LN,66614
234-9566
52 M 1643 77 P
MEGIBOW, ALAN D, TOPEKA PSYCHIATRIC CTR,66601
233-7138
37 M 3503 64 CHP
MEIDINGER, RICHARD, 310 MED ARTS BLDG,66604
234-3451
39 M 1902 65 OR
MENNINGER, KARL A, BOX 829,66601
273-7500
93 M 2401 17 P
MENNINGER, ROBERT G, 234 KANSAS,66603
232-7214
22 M 3545 52 P
MENNINGER, ROY W, 3617 W 6TH,66601
273-7500
26 M 3520 51 P
MENNINGER, W WALTER, THE MENNINGER FO,66601
273-7500
31 M 3520 57 P
MEYER, O WARREN, 634 MULVANE #100,66606
233-9643
49 M 1902 74 CO
MILLS JR, PHILIP E, 901 GARFIELD,66606
357-6171
36 M 1902 64 N
MOOLIN, HERBERT C, MENNINGER FO,66601
273-7500
13 M 3005 38 P
MDORE, HUGH C, 1500 W TENTH,66606
354-6031
33 M 4812 59 PATH
MORRIS, MERLE O, 1401 W 10TH,66604
234-2877
21 M 1902 45 DBG
MORRISON, MICHAEL R, 800 LINCOLN,66606
233-5101
50 M 1902 76 OBG
MORRDW JR, J TARLTON, 235 WDODLAWN,66606
-
23 M 4804 47 OO
MUELLER, ARNOLD V, 901 GARFIELD,66606
354-9591
31 M 3005 57 IM
MYERS, JO ANN, MENNINGER FO,66601
273-7500
28 F 1902 53 P
NABOURS, RICHARD O, 4228 W 29TH ST TERR,66614
272-7190
27 M 1902 54 FP
NANCE, JOEL, MENNINGER FDUNDATION,66601
273-7500
42 M 3546 72 P
NICE, G WILLIAM, 112 MED ARTS BLDG EAST,66604
235-8090
22 M 1902 46 IM
NICHOLS, JEFF S, 1412 W SIXTH,66606
232-6950
44 M 1902 75 PYM
NORTHWAY, DANIEL P, 1200 COLLINS,66604
267-0150
42 M 1102 74 P
NOVOTNY, PETER C, MENNINGER FO,66601
273-7500
30 M 15407 55 P
O'NEIL, ROBERT H, 901 GARFIELD,66606
354-9591
20 M 1902 45 IM
OBDURN, RDBERT L, 1150 OAKLEY,66604
273-7500
19 M 2802 50 P

PALMBERG, KENT E, 901 GARFIELD,66606
354-9591
49 M 1902 74 IM
PARMAN, ROBERT D, 904 MULVANE,66606
232-8224
27 M 1902 S4 PO
PARULKAR, DEEPAK S, 631 HORNE SUITE 120,66606
232-8761
49 M 49S17 73 ANES
PATEL, VINOO, 655 WESTCHESTER RO,66606
3S7-6171
47 M 49531 69 N
PATINO, EDGAR, MENNINGER FO,66601
273-7S00
41 M 26406 66 P
PATRICK, FRED EDWARD, 904 MULVANE,66606
232-8224
45 M 1902 71 PO
PAYNE, ROBERT R, 200 CONTINENTAL MED BLDG,66606
233-7491
29 M 1902 55 ORS
PENN, GEORGE M, VETERANS MEDICAL CENTER,66622
272-3111
30 M 4802 58 P
PEROUE II, W LANG, 631 HORNE #400,66606
3S4-9504
49 M 1902 74 GS
PETERSON, DEAN L, 303 MED ARTS BLDG WEST,66604
235-3451
24 M 1902 54 ANES
PETERSON, ROBERT L, STORMONT-VAIL EMERGENCY,66606
3S4-6108
36 M 1902 62 EM
PETERSON, VERNON J, 310 MED ARTS BLDG,66604
234-3451
42 M S12 68 R
PETRIK, EDWIN L, MEMORIAL HOSPITAL,66607
354-S100
35 M 1902 64 IM
PETTERSON, DENNIS CRAIG, 310 MEDICAL ARTS BLDG,66604
234-34S1
49 M 1902 74 R
PFUETZE, ROBERT E, 209 MED ARTS BLDG EAST,66604
232-3332
09 M 1902 35 OBG
PIERCE, CHARLES F, 918 W 10TH,66604
235-6282
24 M 4101 S1 OBG
PIERCE, DONALD R, 307 MED ARTS BLDG EAST,66604
235-2226
23 M S101 49 FP
POLLY, RICHARD E, 631 HORNE ST,66606
357-0301
42 M 1803 68 ORS
PORTER, ROBERT O, 901 GARFIELD,66606
3S4-9S91
41 M 2802 67 IM
POWELL, WILLIAM R, 833 GARFIELD,66606
233-8941
30 M 1902 54 GS
POWELL II, BENSON M, 400 CONTINENTAL MED BLDG,66606
3S4-9504
26 M 1606 49 TS
PRESTON, RALPH R, 1710 WEST 10TH,66604
234-2624
19 M 1902 44 OPH
PROKOP, BRADFORD S, 900 WASHBURN,66604
233-3900
32 M 1606 57 OPH
PYLE, LUCIEN R, 3139 CANTERBURY LN,66604
-
01 M 1601 28 OBG
RAINBOW-EARHART, KATHRYN A, 2916 KENTUCKY,66605
233-1730
21 F 4707 48 P
RAJU, A S PAOMA, 918 W 10TH,66604
234-3447
39 M 49509 61 TS
RAMSEY, BARTLETT W, 904 MULVANE,66606
232-8224
25 M 1902 50 PO
RANSOELL, EDGAR C, 800 LINCOLN,66606
233-S101
41 M 3005 66 OBG
RANSOM, JAMES H, MEDICAL PLAZA BLDG,66604
234-2663
36 M 1803 62 A
REINKING, VICTOR E, 918 WEST 10TH,66604
233-S084
26 M 1902 51 IM
REYMOND, RALPH O, S-F CAP REG RADIOTHER CTR,66606
234-3451
37 M 2301 67 R

RICCI, ROBERT LAWLER, 901 GARFIELD,66606
354-9591
50 M 1902 75 IM
RICH, JOSEPH E, 634 MULVANE SUITE 104,66606
233-1979
47 M 40921 74 OBG
RICHARDSON, J M, 634 SW MULVANE,66606
232-7762
47 M 1611 74 IM
RIEDEL, ROBERT H, 6731 SW ALYESBURY RO,66610
23S-0011
04 M 2802 28 PH
ROBERTS, WARREN E, PO BOX 4047,66604
272-5797
2S M 1902 57 FP
ROBINSON, DAVID B, 800 LINCOLN,66606
233-5101
47 M 1902 73 OBG
ROEDER, ROBERT E, 901 GARFIELD,66606
3S4-9591
40 M 1902 67 IM
ROEMBACH, JEANINE L, BOX 829,66601
273-7500
52 F 1902 7S CHP
ROSS, JACK L, MENNINGER FO,66601
273-7500
32 M 4812 56 P
ROTERT, LARRY, 301 MEDICAL PLAZA BLDG,66604
233-4256
38 M 300S 66 U
ROY, WILLIAM R, 634 MULVANE STE 104,66606
233-1979
26 M 1606 48 AOM
RUCKER, CLEMENS, 1205 W 29TH,66611
-
86 M 2822 05 OO
RUNNELS, JOHN B, 901 GARFIELD,66606
3S7-6171
3S M 1902 61 NS
RUPP, RICHARD J, 901 GARFIELD,66606
354-9S91
42 M 3841 68 CO
SANCHEZ, ROGELIO, 1516 W 6TH ST,66606
232-100S
31 M 64901 64 U
SARGENT, JOSEPH D, MENNINGER FO,66601
273-7500
32 M 2S01 58 IM
SAYLOR, EDWARD H, 918 WEST 10TH,66604
233-3362
39 M 1902 65 PO
SAYLOR, LESLIE L, 918 WEST 10TH,66604
-
07 M 1606 35 OO
SAYLOR, MARK, 918 WEST 10TH,66604
234-3211
37 M 1902 66 GS
SAYLOR, STEPHEN, 631 HORNE SUITE 340,66606
232-9394
47 M 1902 73 FP
SCAMMAN, W WIKE, PO BOX C-50,66601
234-8621
32 M 4705 57 PATH
SCHLOESSER, HARVEY L, 918 MERCHANTS NATL BK,66612
235-3184
21 M 3901 51 P
SCHLOESSER, PATRICIA T, 1914 WARNER CT,66604
862-9360
24 F 3901 49 PD
SCHRAM, PETER CHARLES, PO BOX 2428,66601
273-7S00
39 M 2S07 69 P
SEGERSON, JOHN A, 901 GARFIELD,66606
357-6171
18 M 3545 43 N
SEHOEV, JDAN, MEMORIAL HOSP,66607
3S4-5100
40 F 6101 63 FP
SETTLE SR, RUSSELL O, 2320 CHELSEA DRIVE,66614
-
04 M 1902 29 OO
SHAW, JOSEPH L, MED ARTS BLDG #204,66604
235-6221
34 M 511 60 ORS
SHEAFOR, DOUGLAS, 925 WESTERN,66606
233-7138
34 M 1902 60 P
SHELTON, STEPHEN E, 92S WESTERN,66606
233-7138
35 M 702 61 P
SHERWOOD JR, CLARENCE E, CONTINENTAL MED BLDG,66606
354-9504
22 M 702 53 GS

SHEU, W ERIC, 1001 HORNE #303,66604
-
43 M 38505 67 ANES
SIMPSON, WILLIAM S, MENNINGER FOUNDATION,66601
273-7500
24 M 6001 48 P
SISK, PHILLIP B, 310 MED ARTS BLDG WEST,66604
234-3451
32 M 1803 56 R
SMITH, LEO A, 4240 EMLAND DR #8 FONTBLU,66606
-
08 M 3006 33 OO
SNARR, JACK W, MED ARTS BLDG W #310,66604
234-3451
41 M 6201 65 DR
SCHN, YUNGGYO, 6018 SW 24TH TERR,66614
-
40 M 58304 66 ANES
SPEARMAN, JESSE L, MED ARTS BLDG ROOM 24,66604
234-2879
20 M 1902 54 OBG
SPENCER, MILLARD C, 310 MED ARTS BUILDING,66604
234-3451
28 M 1902 55 R
SPENCER, WAYNE E, 103 MED ARTS BLDG EAST,66604
233-9686
38 M 1902 64 GE
STEIN, JOSEPH M, 901 GARFIELD,66606
357-6171
24 M 3519 47 N
STEINER, FREDERICK T, BOX 829,66601
273-7500
50 M 5606 76 IM
STOCK, KARL W, 2740 BURLINGAME RD,66611
-
13 M 2834 37 OO
SUFI, M ASHRAF, 1001 HORNE #111,66604
354-8518
43 M 70402 68 IM
SUTTON, RICHARD O, 1706 W TENTH,66604
235-2311
38 M 4706 67 ORS
SWOGGER JR, GLENN, MENNINGER FD,66601
273-7500
35 M 3806 60 P
TAPPEN, DANIEL L, 2849 MACVICAR,66611
-
16 M 1902 41 OO
TARGOWNIK, KARL K, 1218 W TENTH,66604
296-7281
15 M 40710 49 P
TARNOFF, GERALD M, BOX 829,66601
273-7500
48 M 1642 73 P
TARNOWER, WILLIAM, MENNINGER CLINIC,66601
273-7500
21 M 4802 48 P
TEMPERO, STEPHEN J, 310 MED ARTS BLDG,66604
234-3451
42 M 1606 67 R
THOMS, NORMAN W, 40 MED ARTS BLDG,66604
233-1710
34 M 2501 59 TS
THURSTON, DAVID E, 200 CONTINENTAL BLDG,66606
233-7491
29 M 1902 55 ORS
TIETZE, DENNIS O, 634 MULVANE STE 402,66606
235-9545
50 M 1902 78 FP
TOTH, JOHN ROY, 2115 W TENTH,66604
232-3330
49 M 1902 71 FP
TOZER, RICHARD C, 1447 OAKLEY,66604
-
19 M 4102 45 OO
TRAVIS, JOHN W, S-F CAP REG RADIOTHER CTR,66606
295-8000
29 M 1606 55 R
TREES, CLYDE B, 3700 HUNTOON,66604
-
09 M 2401 33 OO
TREGER, NEWMAN V, 1704 W 10TH,66604
354-8761
16 M 1902 40 IM
TUCKER, VIRGINIA L, BUREAU OF MAT & CHILD HLT,66620
862-9360
30 F 1902 57 PD
TWEMLOW, STUART W, 707 QUINCY SUITE 208,66603
233-1607
41 M 67101 73 P
UHR, NATHANIEL, MENNINGER FOUNDATION,66601
273-7500
00 M 3519 21 IM

VAN SICKLE, GREGGORY J, 634 MULVANE,66606
233-8508
49 M 1606 75 PD
VANDE GARDE, LARRY D, 800 LINCOLN,66606
233-5101
41 M 1803 66 OBG
VOGEL, STANLEY J, 901 GARFIELD,66606
354-9591
44 M 2802 70 ONC
WADE III, WILLIAM E, D.D., PO BOX 1531,66601
272-3111
53 M 3979 80 FP
WALLACE, LEO F, 5500 W 24TH,66614
-
17 M 1902 41 OO
WALLS, WILLIAM J, 310 MED ARTS BLDG,66604
354-6171
39 M 2834 66 DR
WALZ, ROYCE C, 1710 W 10TH SUITE 205,66604
234-2676
27 M 15407 60 P
WANLESS, KIRK M, 1424 W EIGHTH,66606
232-8188
44 M 2803 74 OTO
WARD, HOWARD N, 901 GARFIELD,66606
354-9591
37 M 1606 62 HEM
WARE, LUCILE M, MENNINGER FOUNDATION,66601
273-7500
29 F 3501 53 P
WARRICK, DAVID ALAN, 600 MADISON,66607
354-5275
49 M 3843 76 IM
WATERS, DALE A, 1001 HORNE #40,66604
233-1710
41 M 5605 67 CDS
WEAVER, WALTER D, 900 WASHBURN ST,66606
233-3636
41 M 1902 69 OPH
WEBER, DARRELL J, 1710 W 10TH,66604
233-2305
16 M 1902 44 FP
WILCOX, DONALD E, 6700 S TOPEKA AVE,66620
296-3782
24 M 1902 55 PH
WILSON, MARVIN H, 1516 SW SIXTH,66606
233-1747
38 M 1003 64 GS
WINER, RICHARD S, 5813 SW 22ND TERR #4,66614
234-9566
55 M 1902 80 P
WING, NANCY J, 700 HARRISON,66603
295-3175
30 F 5104 56 AOM
WONG, NORMUND, MENNINGER FD,66601
273-7500
34 M 502 59 P
WOOD, EDWARD RUSSELL, 901 GARFIELD,66606
354-9591
49 M 1902 71 IM
WOODS, ROBERT P, 901 GARFIELD,66606
357-6171
14 M 6701 40 N
YEH, ROBERT M, 2315 W 34TH,66611
235-3451
47 M 24405 73 ANES
YORKE JR, CRAIG H, 901 GARFIELD,66606
357-6171
48 M 2401 74 NS
YOUNG, PAUL E, TOPEKA MEDICAL CENTER,66604
233-4927
42 M 2407 75 OPH
YOUNG, THEODORE E, 107 MED ARTS BLDG WEST,66604
232-0576
22 M 2307 46 PD
ZACHARIAS, DAVID LLOYD, 1500 W TENTH,66606
354-6870
26 M 1902 53 PATH
ZIMMERMAN, WILLIAM H, CONTINENTAL BLDG,66606
232-4377
20 M 3006 52 GS

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NYBERG, FREDRIK F, ROUTE 1,67144
-
22 M 2101 46 OO

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WERNER, WILLARD F, ,67879
376-4251
24 M 1902 52 FP

TROY—913
(Northeast Kansas Society)

MASTERSON, MELVIN LEROY, 210 S MAIN,66087
985-2211
23 M 4901 48 R

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(Southwest Kansas Society)

BREWER, MARSHALL A, PO BOX 687,67880
356-1261
19 M 1902 46 FP
RAMCHANDANI, A P, 301 E GRANT,67880
356-2432
39 M 49530 74 FP
TILLOTSON, DON R, BOX 687,67880
356-1261
32 M 1902 65 FP

VALLEY CENTER—316
(Sedgwick County Society)

DANIELS, ROBERT M, BOX 128,67147
262-6262
24 M 1902 54 FP
WILSON, ROBERT L, RR 1,67147
685-2563
30 M 1902 57 EM

WAKEENEY—913
(Central Kansas Society)

HAMILTON, JAMES J, MEDICAL CENTER,67672
743-2124
30 M 1902 55 FP
JACOBSON, JOEL A, 323 RUSSELL,67672
743-2124
42 M 74811 80 GP

WAMEGO—913
(Pottawatomie County Society)

BORGENDALE, LLEWELLYN V, 1601 SUNSET,66547
456-2291
29 M 1902 60 FP
BRADEN, BILL L, 507 ELM,66547
456-2291
31 M 1902 60 FP
CLARK, LAURENCE A, 507 ELM,66547
456-2291
12 M 1902 42 FP
WELCH, MAURA S, BOX 149,66547
456-9202
50 F 1902 75 OBG
WIGGLESWORTH, ANNE, HWY 24 PO BOX 149,66547
456-9202
40 F 66547 75 OBG

WASHINGTON—913
(Northeast Kansas Society)

BITZER, DONALD A, 115 W 3RD,66968

03 M 3005 26 OO
HODGSON, DAVID K, 107 E THIRD,66945
325-2259
49 M 1902 78 FP
MAGEE, LAWRENCE M, 107 E THIRD,66968
325-2240
52 M 1902 77 FP

WATHENA—913
(Northeast Kansas Society)

PETERSON JR, EVAN A, 324 ST JOSEPH ST,66090
989-3122
24 M 1803 55 FP

WELLINGTON—316
(Tri-County Society)

ANDERSON, LARRY R, 1323 NORTH A,67152
326-3301
43 M 1902 73 FP
COLE, WARD M, 110 N JEFFERSON,67152
326-7221
08 M 1902 36 FP
NALOOZA JR, FAUSTINO M, 1323 NORTH A,67152
326-8171
M 74801 64 GS
PERAZA, HERNANDO, 33 CRESTWAY,67152
326-8070
28 M 26404 56 R
WEIGAND, JOEL T, 1323 NORTH A,67152
326-3301
43 M 1902 70 FP

WESTMORELAND—913
(Pottawatomie County Society)

DECHAIRO, THOMAS, DECHAIRO HOSP,66549
457-3311
13 M 1902 36 FP

WICHITA—316
(Sedgwick County Society)

ABBAS, DILAWER H, 841 S HILLSIDE,67211
685-1111
45 M 70402 71 N
ACEVEDO, ALFREDO, 959 N EMPORIA SUITE 205,67214
265-4701
40 M 73701 59 COTS
AGUSTIN, CONRADO M, 1035 N EMPORIA STE 165,67214
267-3389
38 M 74807 62 OBG
AHLSTRAND, RICHARD A, 3243 E MURDOCK SUITE 104,67208
685-2711
41 M 3005 67 R
ALDERETY, NEIL, 3243 E MURDOCK STE 400,67208
686-7351
46 M 64914 75 P
ALEXANDER, ELIZABETH, 3243 E MURDOCK SUITE 300,67208
685-8231
46 F 1902 77 FP
ALFONSO, MANUEL, 3244 E DOUGLAS,67208
689-9445
37 M 84710 66 ANES
ALLEN, PHILLIP M, WESLEY MEDICAL CENTER,67214
688-2838
27 M 2401 54 PATH
ALMONTE, PRISCILLA C, 303 S HILLSIDE,67211
684-7251
44 F 74801 67 ANES
ALMONTE, RODOLFO O, 1431 BLUFFVIEW DR STE 217,67218
686-3791
39 M 74801 64 OBG
AMMAR, ALEX O, 818 N EMPORIA SUITE 200,67214
263-0296
51 M 5101 76 GPVS
ANDERSON, EUGENE G, SUTTON PLACE,67202
265-8619
19 M 1902 44 OBG
ARGOSINO, RODOLFO, 1148 S HILLSIDE,67211
683-6506
40 M 74801 63 GS
ARTZ, TYRONE O, 1125 N TOPEKA,67214
267-0362
41 M 1803 67 ORS
ASHMORE, ARTHUR L, 811 N MISSION,67206

05 M 1902 32 TS
AUNINS, JOHN, 4853 HEMLOCK,67216
524-6805
28 M 4706 56 FP

BACKES, DAVID J, 851 N HILLSIDE,67214
635-8321
48 M 1720 77 U
BAILEY, DONALD C, 3243 E MURDOCK,67208
685-1491
37 M 3901 65 ORS
BAMMEL, BRUCE, 3244 E DOUGLAS,67208
689-9234
52 M 2507 78 OBG
BARBA, ESTRELLA G, 8309 BROOKHOLLOW LANE,67206
264-2301
41 F 74802 66 P
BARBA JR, ANTONIO P, 8309 BROOKHOLLOW LANE,67206
264-2301
34 M 74807 62 OBG
BARKER, BENJAMIN W, 8015 WILLOWBROOK,67207

18 M 1902 51 OO
BARKER, PATSY, 818 N EMPORIA SUITE 303,67214
265-3774
49 F 64914 75 PD
BARNES, JOE L, 1805 GRIFFITH,67207
689-8070
54 M 1902 82 FP
BARNETT, ARNOLD M, 5111 E 21ST,67208
685-2561
32 M 83601 54 N
BARTAL, ELY, 818 N EMPORIA SUITE 306,67214
262-7598
45 M 39607 70 ORS
BARTLETT, WAYNE C, 13 HAMPTON RD,67207
689-9124
07 M 1601 31 GS
BASS II, ORAL E, 851 N HILLSIDE,67214
685-1371
40 M 2803 71 U
BASSELL, G M, 3243 E MURDOCK SUITE 401,67208
686-7327
46 M 14303 73 ANES
BATES, MICHAEL D, 2703 EAST CENTRAL,67214
685-1277
48 M 3005 74 OBG
BATTISTE, CYNTHIA ELAINE, 3333 E CENTRAL SUITE 533,67208
681-2021
47 F 1606 73 PD
BAUMAN, M LEON, 1629 UNIVERSITY,67213
-
01 M 1902 44 DD
BAUMANN, PAUL A, 3333 E CENTRAL,67208
685-1291
32 M 5605 57 R
BEAVER, JAMES L, 245 COURTLEIGH DR,67218
-
09 M 2101 35 OO
BEBAK, DONALD M, 2322 E CENTRAL,67214
263-6186
32 M 3515 58 ANES
BEBER, JORGE H, 2330 N RIVER #821,67220
-
54 M 42901 78
BECK, CHARLES W, 1148 S HILLSIDE SUITE 10,67211
685-8262
46 M 301 72 IM
BECKER, KARL E, 1201 N RIVER BLVD,67203
268-6151
43 M 2307 69 ANES
BENZ, LAURIE J, VETERANS HOSP,67218
685-2221
39 F 4107 65 IM
BETHEL, CHANDLER S, 5107 E 21ST ST,67208
682-6559
34 M 1902 59 IM
BHARGAVA, BAIKUNTH N, 3243 E MURDOCK SUITE 102,67208
682-4523
37 M 49530 63 U
BIERMANN, HENRY J, 425 E MURDOCK,67214
265-6287
27 M 3006 52 GS
BIERMANN, WILLIAM J, 1435 LIEUNETT,67203

04 M 3006 29 OD
BINGAMAN, ROBERT W, 7111 E 21ST,67206
682-1053
47 M 3901 72 GS
BINYON, KERNIE W, 4618 E CENTRAL,67208
684-2819
24 M 1902 56 FP
BLAYLOCK, HOYT C, 835 N HILLSIDE,67214
685-4395
21 M 1902 45 D
BLOOD, MARY J, 647 N HILLSIDE,67214
682-4559
13 F 1902 49 PD

BLOOM, RODNEY LAMONT, 2141 LORI LANE,67207

54 M 1902 79 IM
BLOXHAM, THOMAS J, 3244 E DOUGLAS,67208
689-9215
50 M 1803 75 PUD
BOND, ROGER C, 3243 E MURDOCK,67208
684-0251
40 M 5606 67 CD
BOWMAN, ANTHONY J, 202 N ROCK RD #1706,67206
-
59 M 64914 80
BOYD, Z REX, 120 S MAIZE RD #12,67209
268-5000
26 M 3005 52 FP
BOYLE, HUGH H, 424 N WOODLAWN,67208
686-2193
33 M 3806 60 PATH
BRAKE, DAVID, 3243 E MURDOCK SUITE 104,67208
685-2711
43 M 702 68 R
BRAUN, KENNETH, 1431 S BLUFFVIEW,67218
683-4688
47 M 3519 72 OPH
BRAUN, THOMAS G, 3243 E MURDOCK #601,67208
685-2377
35 M 6001 61 N
BRAUN III, WILLIAM T, 3243 E MURDOCK SUITE 104,67208
685-2711
37 M 2802 61 R
BRECKBILL, DAVID L, 3333 EAST CENTRAL #214,67208
685-1291
38 M 1902 64 R
BRINTON, E HOLMES, 3244 E DOUGLAS,67208
689-9124
46 M 2101 70 GS
BRINTON, EDWARD S, 329 NORTH TERRACE DR,67208

15 M 1611 41 OO
BRITO, RAUL E, 3243 E MURDOCK,67208
682-4523
32 M 31901 59 U
BROSIUS, FRANK C, 3243 E MURDOCK,67208
684-0251
25 M 1902 49 IM
BROWN, DAVID J, 425 EAST MURDOCK,67214
265-6287
45 M 1902 71 GS
BROWN, MICHAEL P, 345 N HILLSIDE,67214
683-6766
51 M 3007 77 OBG
BROWN, RANDALL J, 8800 E HARRY SUITE 408,67207
-
55 M 1902 81 FP
BROWN, ROBERT L, 5025 E KELLOGG,67218
682-1534
21 M 1902 49 FP
BROWN, RONALD C, 3243 E MURDOCK,67208
685-8231
47 M 2803 73 FP
BROWN, RONALD L, 1128 S CLIFTON,67218
684-7251
45 M 3901 71 ANES
BROWN, VAL J, 1802 N HYDRAULIC,67214
265-1461
24 M 1003 47 FP
BROWN JR, VAL J, 1802 N HYDRAULIC,67214
265-1461
53 M 1902 79 IM
BROWNING, WILLIAM H, 851 N HILLSIDE,67214
685-1371
16 M 1902 43 U
BRUNER JR, KENNETH W, ST FRANCIS HOSPITAL,67214
268-5414
44 M 2401 70 PATH
BUBECK, RALPH W, 3244 E DOUGLAS,67208
689-9396
36 M 1803 62 IM
BUCK JR, BEN H, 1431 S BLUFFVIEW STE 217,67218
684-1048
17 M 2834 43 TS
BUCKRIDAN, LATIF, 7232 CHELSEA,67206
684-7251
31 M 6501 62 ANES
BURNEY, WILLIAM W, 1755 N MADISON,67214
264-8311
17 M 1902 52 FP
BURNEY II, WILLIAM W, 1755 N MADISON,67214
264-8311
50 M 4707 76 IM
BURPEE, JAMES F, 851 N HILLSIDE,67214
685-1371
39 M 5605 66 U

BUTH, DENNIS K, 2916 EAST CENTRAL,67214
684-5243
45 M 1902 72 IM
BUTIN, J WALKER, 3244 E DOUGLAS,67208
689-9477
23 M 1902 47 IM
BUTLER, DORIS C, 1148 S HILLSIDE,67211
684-2329
48 F 1902 75 FP
BYRNE, JAMES PERRY, 818 N EMPORIA SUITE 200,67214
263-0296
42 M 2101 68 TS
CALIENDO JR, DANIEL J, WESLEY MED CENTER,67214
685-2563
41 M 1902 67 EM
CANNON, MICHAEL W, 1035 N EMPORIA,67214
588-6029
50 M 1902 75 DN
CAPPER, STANLEY L, 3244 E DOUGLAS,67208
689-9206
37 M 1803 67 D
CARLSON, TERRY S, 550 N HILLSIDE,67214
688-2820
50 M 3006 77 PATH
CARSON, BERTRAM H, D.O., ST FRANCIS HOSP,67214
268-5050
26 M 2878 64 EM
CARTER, MACK A, 655 N WOODLAWN,67208
684-5158
18 M 1902 50 DPH
CASEY, JOHN J, 3243 E MURDOCK SUITE 604,67208
685-8144
25 M 2604 49 NS
CAUBLE, WILBUR G, 1148 S HILLSIDE SUITE 102,67211
683-1681
12 M 2834 39 GS
CAWLEY, LED P, WESLEY MED CENTER,67214
685-2151
22 M 3901 52 PATH
CHANEY, ERNIE J, ST JOSEPH MED CENTER,67218
685-1111
27 M 1902 56 FP
CHANG, FREDERIC C, 818 N EMPORIA SUITE 200,67214
263-0296
35 M 2401 59 GS
CHAPMAN, JAMES H, PO BOX 2579,67201
265-1684
27 M 4706 63 R
CHARD, FREDERICK H, 3244 E DOUGLAS,67208
689-9129
15 M 5605 39 D
CHI, IL-SUNG, 3243 E MURDOCK SUITE 401,67208
686-7327
41 M 58302 67 ANES
CHO, SECHIN, UKSM - WICHITA,67214
268-8302
47 M 58302 71 PD
CHOPRA, RAMAN, 3333 E CENTRAL #201,67208
685-5271
52 M 49536 76 PD
CLARK, COURTNEY, 303 S HILLSIDE,67211
684-7251
30 M 1902 56 ANES
CLARK, ROBERT G, 1023 N TERRACE,67208
688-2688
53 M 1902 78 GS
CLIFTON, H DAVID, 3600 E HARRY,67218
685-1111
41 M 401 65 R
CLINE, BYRON W, 550 N LORRAINE,67214
685-0559
51 M 4802 77 DBG
COHEN, JUSTIN THOMAS, 655 N WOODLAWN,67208
684-5158
47 M 2803 74 DPH
COHLMIA, JERRY B, 818 N EMPORIA SUITE 310,67214
263-5891
43 M 1902 70 IM
COLEMAN, THOMAS J, 959 N EMPORIA,67214
265-0749
18 M 3545 51 IM
COLLIER, HAROLD W, 144 N OLIVER SUITE 325,67208
683-5008
45 M 1902 71 ANES
CONCEPCION JR, EUGENIO S, 1431 S BLUFFVIEW STE 217,67218
684-1048
39 M 74802 64 CD
CONRARDY, PETER A, 1201 N RIVER BLVD,67203
686-7327
42 M 515 69 ANES
COOK, DONALD RAY, 315 N HILLSIDE,67214
686-3391
42 M 2012 71 FP

COOK, G EDWARD, ST JOSEPH HOSPITAL,67218
685-1111
42 M 401 67 R
COOPER, MICHAEL K, 1201 N RIVER BLVD,67203
263-1574
54 M 1902 79 ANES
COSSMAN, F PRICE, 851 N HILLSIDE,67214
685-1371
28 M 1902 57 U
COWLES, GORDON T, 3333 E CENTRAL,67208
683-2661
32 M 1902 58 DBG
CRANE, DAVID D, 929 N ST FRANCIS,67214
262-6211
34 M 2501 60 PATH
CRONIN, DONALD J, 3244 E DOUGLAS,67208
689-9227
16 M 2604 40 ENT
CROW, ERNEST W, 3243 E MURDOCK,67208
684-0252
20 M 1902 44 CD
CROWLEY, EDWARD X, 345 N HILLSIDE,67214
682-4519
14 M 1643 39 GYN
CUMMINGS, RICHARD J, 427 N HILLSIDE,67214
686-6608
32 M 1902 57 DTD
DAKHIL, SHAKER R, 1035 N EMPORIA SUITE 210,67214
262-4467
50 M 60501 75 ONC
DARRAH, CAROL J N, 3333 E CENTRAL SUITE 613,67208
685-9289
49 F 1902 74 R
DAVIDSON, HARRY T, 556 N BRADVIEW,67208

87 M 3802 11 DD
DAVIS, PAUL H, 7111 E 21ST,67206
684-2851
47 M 3901 72 FP
DAVIS, RONALD B, 1148 S HILLSIDE,67211
685-2153
46 M 1902 72 FP
DAY, HOWARD, 818 N EMPORIA SUITE 310,67214
263-5891
48 M 1902 74 NEP
DE BAKKER, JAN B, 1035 N EMPORIA,67214
263-4903
25 M 5104 59 GS
DE HART, ARTHUR DONIVA, 2703 E CENTRAL,67214
685-1277
50 M 4804 77 DBG
DEJONG, DAVID C, 550 WEST SHORE DR,67209
268-5424
33 M 2501 59 PATH
DEMOSS, ELEANOR P, 3333 E CENTRAL SUITE 407,67208
682-5591
42 F 74802 66 PD
DENNING, DALE P, 631 N CRESTWAY,67208
684-2014
56 M 1902 82
DIACON, JAMES L, 2029, N WOODLAWN #710, 67208
326-2111
24 M 3901 52 FP
DIAZ, DOLORES M, 2579 GREENLEAF CT,67226
686-7327
51 F 64902 75 ANES
DIAZ, SALVADOR F, 3243 E MURDOCK SUITE 401,67208
686-7327
45 M 64902 73 ANES
DIRKSEN, HANS C, 550 N HILLSIDE,67214
688-2360
47 M 6001 71 NED
DOEBLIN, P LAURENCE, 3333 E CENTRAL SUITE 214,67208
685-1291
40 M 1002 73 R
DOLAN JR, PHILIP JARVIS, 3244 E DOUGLAS,67208
689-9477
47 M 2105 73 GE
DONATELLE, EDWARD P, UKSM WICHITA,67214
268-8221
22 M 2604 50 FP
DONLEY, JAMES L, 3101 E NINTH,67208
684-0201
46 M 1902 72 P
DONNELL, JAMES M, 2320 E CENTRAL,67214
264-8010
28 M 1902 55 FP
DORNBOS, J FRED, 929 N ST FRANCIS,67214
262-6211
28 M 1902 57 R
DOUTHIT, DOUGLAS D, 550 N LORRAINE, 67214
685-0599
53 M 79 OBG

DRAKE, RALPH L, 4422 E 3RD,67208

99 M 4102 26 DD
DRANGE, ROBERT K, 3333 E CENTRAL STE 214,67208
685-1291
52 M 1902 77 DR
DRAZEK, GEORGE, 3244 E DOUGLAS,67208
689-9111
50 M 3506 76 DPH
DREVETS, CURTIS C, 3244 E DOUGLAS,67208
689-9178
30 M 1902 56 IM
DUICK, GREGORY, 1035 NORTH EMPORIA #130,67214
263-3271
46 M 1643 72 CO
DURAND, ANTONIO C, 959 N EMPORIA,67214
263-7893
29 M 74807 56 U
DYCK, CORA E, 341 S CRESTWAY,67218

00 F 1902 26 OO
DYER, VERNON E, 3244 EAST DOUGLAS,67208
262-6202
36 M 301 72 DBG
ECKERT, WILLIAM G, ST FRANCIS LAB,67214
268-5470
26 M 3519 52 PATH
EDWARDS, MANIS C, 3333 E CENTRAL,67208
683-2661
33 M 3005 58 DBG
EGELHOF, RICHARD H, 1035 N EMPORIA SUITE 290,67214
263-1299
45 M 1902 73 FP
ELLIS, HARVEY D, 6611 E CENTRAL,67206
683-1022
24 M 1902 55 GS
ELLIS, JOSEPH G, 3244 E DOUGLAS,67208
689-9132
25 M 511 62 IM
ELNEN, WALTER T, 460 N TERR DR,67208
682-5671
03 M 1643 32 GS
ENDOH, ROLLAND, 315 N HILLSIDE,67214
681-0423
49 M 64914 76 FP
ERKEN, RONALD V, WICHITA PSYCHIATRIC CTR,67208
684-0201
29 M 2834 56 P
DUNSHEE, CHERYL A, UKSM WICHITA, 67214
268-8378
54 F 1902 79 IM
ERNST, R L, 400 W CENTRAL APT 2504,67203

56 M 3005 82 GS
ESTEP, THOMAS H, 818 N EMPORIA SUITE 200,67214
263-0296
51 M 6002 75 CD
EVANS, FARRIS D, 521 RUTLAND RD,67206

05 M 1902 32 FP
EVANS, GRANT E, 4618 E CENTRAL,67208
682-6556
21 M 4901 46 FP
EVANS, JOHN F, 8542 MT VERNON CT,67207
688-2360
42 M 2803 70 MFM
EVANS, ROGER WILLIAMS, 933 N TOPEKA,67214
263-5889
39 M 1902 64 CD
EYSTER, ROBERT L, 3243 E MURDOCK,67208
685-1491
47 M 3901 73 ORS
FARHA, GEORGE J, 818 N EMPORIA SUITE 200,67214
263-0296
27 M 2101 57 GS
FARHA, S JIM, 818 N EMPORIA SUITE 200,67214
263-0296
31 M 1001 57 TS
FARLEY, JAMES A, ST JOSEPH MEDICAL CENTER,67218
685-1111
50 M 1902 78 PATH
FENDER JR, THOMAS H, 9400 E LINCOLN APT 113,67207
267-8439
25 M 4812 54 DS
FERRELL, DONALD P, ST JOSEPH MEDICAL CTR,67218
685-2371
36 M 3901 63 EM
FERRIS, BRUCE G, 825 N EMPORIA,67214
262-3495
43 M 1902 69 PS
FEUILLE JR, EDMOND G, 212 N HILLSIDE,67214
682-4572
50 M 4802 75 DBG

FIELDS, STEPHEN, D.O., 7200 W 13TH,67212
722-4258
42 M 2878 72 FP
FINLEY, DENNIS R, 1035 N EMPORIA,67214
262-7429
36 M 1606 62 ORS
FISHER, JAMES B, 141 S OLD MANOR,67218

09 M 1902 36 DD
FISHER, RAY F, 3243 E MURDOCK SUITE 500,67208
684-0251
49 M 1902 74 IM
FITZGERALD, EDWARD J, 3600 E HARRY,67218
685-1111
22 M 3006 50 R
FITZIG, SANFORD, 3244 E DOUGLAS,67208
689-9344
46 M 4102 72 U
FLEMING, FORNEY W, 3243 E MURDOCK #200,67208
685-1491
43 M 4802 69 ORS
FLOWERS JR, CLELL B, 855 N HILLSIDE,67214
685-1381
22 M 1902 55 FP
FORD, CHARLES R, 232 S MAIZE RD,67209
722-0568
38 M 1902 63 DPH
FOWLER, ROBERT J, 3244 E DOUGLAS,67208
689-9236
37 M 2802 63 IM
FRANCIS, NORTON L, 55 VIA ROMA,67230

10 M 3005 35 ENT
FRANCISCO, DAN A, 3243 MURDOCK SUITE 500,67208
684-0251
40 M 1803 75 CD
FRANCISCO, LINDA L, 818 N EMPORIA SUITE 310,67214
263-5891
47 F 1803 74 NEP
FRASER, CRYSTAL A, 1021, N BATTIN, 67208

56 F 1902 82 P
FRENCH, JAMES, 1144 CLIFTON, 67218
684-5237
53 M 3005 78 GS
FRENCH, JEROME E, MID-KANSAS ENT ASSOC,67211
684-2838
44 M 1103 71 OTO
FRITZ, GEORGE E, WESLEY MED CENTER,67214
685-2151
18 M 2507 43 PATH
FRITZEMEIER, WILLIAM H, 835 N HILLSIDE,67214
685-4395
14 M 1902 41 D
FROMER, JOEL, 2627 E CENTRAL,67214
684-0501
46 M 16501 75 A
FROMM, ARTHUR H, 315 N HILLSIDE,67214
685-2281
37 M 1902 63 FP
FULTON, JOHN K, 3333 E CENTRAL SUITE 801,67208
686-0732
18 M 5605 43 PUO
GALICHIA, JOSEPH P, PO BOX 594,67201
263-3271
42 M 1902 69 CD
GALVAN, ALONSO, 3243 E MURDOCK,67208
684-0251
38 M 64906 64 IM
GENILD, AMANCIO C, 120 LONGFORD CT,67206
685-2371
37 M 74801 61 EM
GENILD, CELESTE A, 3244 EAST DOUGLAS,67208
689-9445
39 F 74801 62 ANES
GEORGE, EARL F, 5107 E 21ST,67208
265-6991
35 M 1902 65 FP
GEORGE, M DON, WICHITA PSYCHIATRIC CL,67208
684-0201
31 M 1001 56 P
GERBER, ALLEN D, 7111 E 21ST,67206
682-1053
48 M 1902 71 GS
GESSLER, DONALD J, 1122 N TOPEKA,67214
265-2876
41 M 1902 67 FP
GILMARTIN, RICHARD C, 3243 E MURDOCK #601,67208
685-2377
32 M 4112 58 PDN
GIVNER, DAVID, 2627 E CENTRAL,67214
684-0501
03 M 2301 29 IM

GOHIL, MAHENDRA N, 1823 RUTLAND,67206
265-1684
28 M 40930 66 OR
GOLOBERG, HERBERT R, 400 WOODLAWN SUITE 101,67208
685-4215
33 M 3508 59 PD
GONZALEZ, HIRAM, 3429 E DOUGLAS,67218
681-1348
20 M 64901 52 P
GOODPASTURE, HEWITT C, 818 N EMPORIA SUITE 305,67214
264-3505
43 M 1902 69 IM
GORDON, JAMES R, 3244 E DOUGLAS, 67208
689-9260
53 M 1611 78 IM
GOYLE, KRISHAN K, 1144 N ST FRANCIS,67214
267-0159
34 M 49529 63 CO
GOYLE, VIMAL, 1150 N ST FRANCIS,67214
267-9906
41 F 49529 65 OBG
GRAUEL, CHARLES W, 8310 CHALET,67207
262-1127
44 M 1902 70 ANES
GRAVES, JACK W, 3244 E DOUGLAS,67208

17 M 1902 42 OO
GRAY, C LUCIEN, 4821 E CENTRAL,67208
684-5171
21 M 1902 45 ENT
GRAY, H TOM, 3610 E ENGLISH 1028,67218

19 M 401 44 OO
GREER, JAMES A, 3244 EAST OOUGLAS,67208
689-9227
43 M 1611 69 OTO
GRIBBLE, ROBERT N, 1035 N EMPORIA #285,67214
264-2401
43 M 1902 69 R
GRILLOT, FLOYO B, 814 S WOODLAWN,67218
684-0243
18 M 1902 51 FP
GROHS, HEINZ K, 550 NORTH HILLSIOE,67214
685-2151
42 M 15407 66 PATH
GRUND, FRANK M, 3244 EAST OOUGLAS,67208
689-9420
47 M 1803 72 IM
GRUSHNYS, ARNOLO, 3244 E DOUGLAS,67208
689-9445
19 M 40721 59 ANES
GSELL, GEORGE F, 32 MISSION RO,67206

07 M 1601 33 OO
GUTHRIE, RICHARD A, KS REG OIABETES CENTER,67214
268-8228
35 M 2803 60 PO
GWINN, OOUGLAS R, 514 N HOLYOKE #204,67208

53 M 2803 80 FP
HABASHY, SHAWKY N F, 1122 N TOPEKA,67214
268-5996
43 M 33004 65 OBG
HAGAN, C THOMAS, 959 N EMPORIA,67214
265-0789
16 M 3006 42 IM
HAGAN, FRANCIS J, PO BOX 1837,67201
262-1057
13 M 3006 39 FP
HAGAN, ROBERT C,3244 E DOUGLAS, 67208
689-9306
52 M 1902 77 GE
HALE, RALPH, 847 S HILLSIDE,67211
684-0295
18 M 1902 46 A
HALL, J ROGER, 3333 E CENTRAL,67208
685-5227
42 M 4802 68 OPH
HALPIN, EOWARO O, 1148 S HILLSIOE,67211
685-9229
33 M 1902 62 OBG
HARMS, EDWIN M, 5623 POLO DR,67208

06 M 3901 34 OO
HARRIS, FRANK H, 1035 N EMPORIA,67214

09 M 1001 39 NP
HARRISON, A BROOKS, 1040 STRATFORD,67206

99 M 1902 25 OO
HARRISON, PAUL BARRY, 3243 EAST MURDOCK,67208
685-6222
49 M 1902 74 GS

HARSTINE, LILLIAN R, 7111 E 21ST,67206
684-2851
51 F 1902 76 IM
HART, DILLIS L, 1152 S CLIFTON,67218
684-2081
36 M 3901 64 GS
HARTLEY, JAMES M, UKSM - WICHITA,67214
685-2387
45 M 2604 71 FP
HARVEY, ROSEMARY B, 635 N MAIN,67203
268-8025
24 F 1902 49 ADM
HASSAN, RIZWAN U, 841 S HILLSIOE,67211
686-2831
47 M 70305 70 N
HAYES, WILLIAM L, 3243 E MURDOCK,67208
684-0251
28 M 1902 53 CD
HAYNES, OEBORAH G, 1131 S CLIFTON,67218
685-4354
54 F 1902 79 FP
HAYS, THOMAS H, 7111 E 21ST,67206
684-2851
49 M 1902 75 FP
HENNING, CHARLES E, 320 N HILLSIOE,67214
682-3221
37 M 1902 63 ORS
HERED, JOHN, 959 N EMPORIA SUITE 403,67214
262-3613
41 M 2802 67 N
HERSHBERGER, GROVER, O.O., 1245 N WEST ST,67203
945-6910
47 M 2878 79 GP
HERSHORN, SIMON E, 3333 E CENTRAL,67208
685-1291
22 M 1902 46 R
HIEBERT, ABRAHAM E, 1530 W 13,67203

94 M 2802 25 OO
HINSHAW, ALFRED H, 6110 ONEIDA,67208

07 M 1902 33 OO
HINSHAW, CHARLES T, 256 N BLECKLEY DR,67208

99 M 4705 26 OO
HINSHAW JR, CHARLES T, 3715 E DOUGLAS,67218
684-7784
32 M 1902 58 PATH
HIRATZKA, TOMIHARU, 6 ST CLOUD PLACE,67230

13 M 511 43 OO
HIZON, RAMON R, ST FRANCIS HOSP,67214
262-6211
38 M 74801 62 OR
HODGSON, ROBERT B, 841 NORTH BRDADWAY,67214
263-6131
22 M 1902 47 FP
HOOSON, HERVEY R, 1122 S CLIFTON,67218

03 M 1606 31 OO
HOLDEN, OAVID M, 3243 E MURDOCK SUITE 300,67208
688-3070
38 M 801 63 FP
HOLDEN JR, RAYMOND F, 262 SOUTH BRODKSIDE,67218

10 M 2802 33 DD
HOLLADAY, HARMON M, 327 N RUTAN,67208
681-0431
23 M 1902 52 ANES
HOLMES, JED, 7111 E 21ST,67206
684-2851
53 M 3005 78 FP
HORBELT, DOUGLAS V, WESLEY MEDICAL ARTS BLDG,67208
681-0251
47 M 4802 72 ORG
HOUSHOLDER, OANIEL FAIR, 7705 KILLARNEY CT,67206
268-5910
43 M 1902 70 NM
HOUSHOLDER, MARTHA S, 835 NORTH HILLSIDE,67214
685-4395
46 F 1902 72 O
HOWARO, DONALD O, 959 N EMPORIA,67214
265-7241
11 M 1902 38 OPH
HUEBERT, DEAN A, 5025 E KELLOGG,67218
682-1534
22 M 1902 46 FP
HULL, KENNETH L, 3429 E OOUGLAS,67218
265-7903
38 M 2301 69 P
HULTGREN, MYRON K, 1520 S CLIFTON,67218
685-1381
41 M 1902 68 FP

HUME, JOSEPH W, 5111 E 21ST,67208
685-2223
38 M 1902 69 OBG
HUMMER, LLOYD M, 3244 E DOUGLAS,67208
689-9323
32 M 3901 57 IM
HUND, LARRY R, 3333 E CENTRAL SUITE 710,67208
682-0411
52 M 1902 78 PD
HUSTEAD, ROBERT F, 427 N HILLSIDE,67214
683-7200
28 M 801 54 ANES
HYNES, HARRY E, 1035 N EMPORIA,67214
262-4467
35 M 53902 58 HEM
IBARRA, J LUIS, 1035 N EMPORIA,67214
262-1853
20 M 64901 46 P
IDBEIS, BADR, 1100 N ST FRANCIS STE 240,67214
267-0159
47 M 87501 72 TS
JACKSON, CATHERINE M, UKSM WICHITA,67214
268-8388
40 F 35207 64 CHP
JACKSON, CHARLES R, 1035 N EMPORIA,67214
263-0812
27 M 1606 53 GS
JAMES, DONALD L, 1301 N WEST,67203
945-5245
42 M 3901 71 OTO
JAMES, VERNON L, 3333 E CENTRAL SUITE 816,67208
685-5326
29 M 3601 55 PD
JAWADI, JAMEELA HUSAIN, 3333 E CENTRAL SUITE 610,67208
686-6659
50 F 49521 73 PD
JAZAYERLI, NABIL, 1152 S CLIFTON,67218
681-2401
44 M 87501 70 CD
JEHAN, SAYED S, 635 N MAIN,67203
268-8306
33 M 70403 59 P
JENNEY, CHARLES B, 818 N EMPORIA SUITE 200,67214
263-0296
34 M 2834 61 GS
JENSEN, DARAN L, 3333 E CENTRAL, 67208
—
52 3005 79 OBG
JESTER, SHELBY L, 250 N PERSHING,67208
263-1574
43 F 4102 74 ANES
JOHNSON, CAROL ANN, 3243 E MURDOCK SUITE 303,67208
688-3070
49 F 1902 77 FP
JOHNSON, GEORGE K, UKSM WICHITA,67214
268-5984
40 M 1205 67 IM
JOHNSON, THOMAS E, 3333 E CENTRAL,67208
685-1291
41 M 1643 67 R
JOSEPH, JAPHET G, 1111 N ST FRANCIS,67214
265-2613
49 M 49531 74 CD
JOST, GARY D, 212 N HILLSIDE,67207
682-4572
51 M 1902 77 GS
JUDILLA JR, FRANCISCO, 2322 EAST CENTRAL,67214
263-6186
44 M 74801 71 ANES
KADISON, HERBERT I, ST FRANCIS HOSP,67214
262-6211
44 M 1611 69 R
KARDATZKE, E STANLEY, 10300 W MAPLE,67209
943-9342
39 M 1720 64 FP
KARDATZKE, JON K, 10300 MAPLE,67209
943-3271
36 M 1720 62 FP
KASHA, ROBERT L, 5025 E KELLOGG,67218
682-1534
11 M 2834 38 GS
KASSEBAUM, KENNETH G, 3243 E MURDOCK SUITE 400,67208
685-6381
34 M 1606 60 CHP
KAUFMAN, EUGENE E, 3243 E MURDOCK,67208
685-1491
30 M 1902 56 ORS
KEENE, GEORGE H, 5025 E KELLOGG,67218
682-1534
20 M 1902 49 GS
KELLER, JAMES P, 1431 S BLUFFVIEW STE 209,67218
685-1284
48 M 1902 74 IM
KELLY, ROBERT W, 550 N LORRAINE,67214
682-6511
46 M 4802 72 OBG
KENDALL, TOM E, 825 N EMPORIA,67214
262-3495
37 M 3901 62 PS
KENDRICK, J GILLERAN, WESLEY MEDICAL CENTER,67214
688-2080
20 M 1902 46 ADM
KENNEDY, GERALD T, 16069 CAVALCADE LANE,67230
684-5243
35 M 1902 61 GE
KHICHA, GYANCHAND J, 818 N EMPORIA SUITE 200,67214
263-0296
37 M 49530 61 TS
KHOURY, GEORGE H, 3333 E CENTRAL SUITE 416,67208
681-2021
32 M 33002 55 PD
KIM, PAIK N, 1035 N EMPORIA,67214
262-4467
33 M 58302 58 ONC
KIMBLE, JAMES A, 3244 E DOUGLAS,67208
689-9316
45 M 702 71 OPH
KIRK JR, E DAVID, 1431 S BLUFFVIEW DRIVE,67218
685-1351
34 M 1902 62 IM
KISER, JOHN L, 3243 E MURDOCK,67208
685-6222
37 M 2802 62 GS
KISER, WILLARD J, 3243 E MURDOCK,67208

05 M 4705 30 GS
KITCHEN, ROBERT R, 3420 E DOUGLAS,67208
685-2355
26 M 1902 52 CHP
KLONTZ, WILLIAM JOSEPH, UKSM WICHITA,67214
268-8388
51 M 1902 76 P
KNAPP, LESLIE E, 302 S CRESTWAY,67218

96 M 1902 25 DO
KNAPP, M ROBERT, 37 VIA RDMA,67230
684-7251
23 M 3519 47 ANES
KNEIDEL, THOMAS W, 732 N TOPEKA,67214
267-1924
40 M 4101 66 ORS
KNIGHT, LAURA C, ST FRANCIS REG MED CTR,67214
268-5922
42 F 502 68 DR
KNIGHT, PHILIP J, 818 N EMPORIA SUITE 200,67214
263-0296
42 M 0502 68 PDS
KOURI, SAMMY H, 3243 E MURDOCK,67208
682-2911
33 M 3901 57 GS
KRAUSE, ROLAND L, 855 N HILLSIDE,67214
685-1381
25 M 1902 53 IM
KREADY, JOHN L, 3243 E MURDOCK SUITE 303,67208
688-3070
48 M 1902 79 FP
KRUPKA, JOHN J, 818 N EMPORIA #201, 67214
263-0348
47 M 1642 73 NS
KUBINA, GLENN RICHARD, MID-KANSAS ENT ASSOC,67211
684-2838
47 M 3840 72 OTO
KURTH, C JOSEPH, 200 S ROCK RD SUITE H,67207
687-6603
10 M 3006 35 NP
KUTILEK, FRANK J, 7200 W 13TH ST SUITE 3,67212
722-4258
30 M 1902 57 FP
LACEY, STEFFAN R, ST FRANCIS HOSP,67214
268-5470
50 M 3005 76 PATH
LAI, JENG Y, 959 N EMPORIA STE 205,67214
265-2613
41 M 38502 67 TS
LANCE JR, JOHN F, 3244 E DOUGLAS,67208
689-9438
20 M 1902 45 DRS
LATIMER, KATHERINE, 3243 E MURDOCK SUITE 401,67208
686-7327
49 F 1205 75 ANES
LAUVER, MARY ANN, 818 N EMPORIA SUITE 303,67214
265-3774
40 F 1902 74 PD
LAWN, CLAUDIA A, 1035 N EMPORIA SUITE 285,67214
264-2401
50 F 1902 75 R

LAWN, RAYMDND A, 715 N MISSIDN RD,67206
683-8991
09 M 2604 35 AM
LAZAR, HARRY, 2627 E CENTRAL,67214

08 M 1611 35 A
LEE, R REX, 6155 E HARRY,67218
685-2306
29 M 3901 55 FP
LEE JR, EDWARD S, 2002 E 17TH ST,67214

09 M 4707 37 FP
LEISY, JERALD W, 3310 E DDUGLAS SUITE 101,67208
681-2937
42 M 1902 68 P
LEITNER, YDRAM 8, 3244 E DDUGLAS,67208
689-9227
53 M 3519 77 OTD
LEVINE, WILLIAM R, UKSM-WICHITA,67214
268-8388
42 M 1902 67 P
LEWIS, LARRY D, 2255 S 8RDADVIEW,67218

54 M 3901 80 FP
LIES, RICHARD 8, 3244 E DDUGLAS,67208
689-9111
42 M 1902 68 RHU
LIN, JDE J, 8630 HUNTINGTDN,67206
268-5420
42 M 24404 69 PATH
LINHARDT, RDNALD D, 3243 E MURDOCK,67208
683-2655
36 M 2803 64 DBG
LITTELL, JAMES A, 1520 S CLIFTDN,67218
685-2371
44 M 1902 71 EM
LITTLE, L GIL8ERT, 122 S WESTFIELD AVE,67209

99 M 401 27 P
LDCKHART, JDSEPH G, 5900 E CENTRAL,67208
684-7239
17 M 4113 43 PD
LDEFFLER, JAMES A, 400 N WDDDLAWN,67208
685-5375
36 M 3841 63 A
LOEWEN, HENRY H, 2142 W 17TH,67203

03 M 1902 35 DD
LOEWEN, WILLIAM C, 10300 W MAPLE,67209
722-6892
41 M 1902 71 FP
LDGAN, GEDFFREY G, 212 N HILLSIDE,67214
682-4572
31 M 14303 56 D8G
LDSEE, JOHN M, 3243 E MURDOCK STE 401,67208
686-7327
51 M 4301 77 ANES
LOVETT, PAUL A, 110 PATTON,67208

09 M 1902 45 DRS
LDW, HARDLD L, 1148 S HILLSIDE,67211
684-2858
18 M 1902 44 FP
LUEKEN, LUEKE 8, 3244 E DDUGLAS,67208
689-9234
23 M 40723 52 D8G
LUELLEN, THOMAS J, 3244 E DOUGLAS,67208
689-9244
17 M 1902 41 IM
LUZZATI, ENZO F, ST FRANCIS HDSPITAL,67214
262-6211
25 M 56119 S0 R
LYNCH, MARY A, 320 N HILLSIDE,67214
682-3221
48 F 1002 77 SM
MADISDN JR, WARD N, 3600 E HARRY,67218
686-9432
37 M 3601 62 PATH
MAGIDSDN, ELLIDT ARTHUR, 116 LDNGFDRD CT,67206
686-2193
43 M 1611 68 PATH
MAILMAN, GERSHDM, ST JDSEPH MED CENTER,67218
685-1111
26 M 3S19 49 ANES
MANNING, ROBERT T, 1001 N MINNEAPDLIS,67214
268-8378
27 M 1902 54 IM
MANSDUR, 8ADIE S, 3243 E MURDDCK SUITE 401,67208
686-7327
45 M 33002 69 ANES
MARSH, HENRY D, 818 N EMPDRIA SUITE 306,67214
262-7598
18 M 1611 43 DRS

MARTIN JR, GLEN E, 2322 E CENTRAL,67214
263-6186
20 M 1902 49 ANES
MARYMDNT JR, JESSE H, WESLEY MED CENTER,67214
685-2151
28 M 351S S4 PATH
MASSEY, ANDREW D, 1001 N MINNEAPDLIS,67214
268-8378
50 M 1902 77 IM
MASTID JR, GEDRGE J, 3243 E MURDOCK,67208
684-S235
25 M 1902 52 GS
MATASSARIN, 8ENJAMIN M, 2916 E CENTRAL,67214
684-5243
20 M 1902 45 IM
MATASSARIN, FREDERICK W, 734 N EMPDRIA,67214
265-2382
15 M 1902 37 U
MAWDSLEY, MICHAEL W, 3333 EAST CENTRAL #610,67208
686-6659
49 M 1902 74 PD
MC NICKLE, GEORGE ANDREW, 222 S RIDGE RD,67209
945-0142
47 M 1902 75 FP
MC8DYLE, MARILEE, 818 N EMPDRIA,67214
263-0296
52 F 1902 77 GS
MCCLANAHAN, WARD A, S105 E 21ST,67208
684-8211
22 M 3005 48 DRS
MCCLELLAN, ERNEST L, 3243 E MURDDCK SUITE 401,67208
686-7327
38 M 4802 70 ANES
MCCDY, CHARLES P, 3333 E CENTRAL,67208
685-7234
17 M 3006 42 DBG
MCDDNDUGH, WILLIAM D, 3244 E DDUGLAS,67208
689-9111
48 M 3305 76 U
MCGUIRE, WILLIAM F, 3333 E CENTRAL,67208
683-5655
17 M 4101 43 PD
MCMULLEN, 8RUCE R, 1122 S CLIFTON,67218
682-5012
53 M 4002 79 IM
MCQUEEN, DAVID ARNDLD, 818 N EMPDRIA,67214
262-7598
47 M 64914 75 DRS
MEADDWS, DONALD C, 3333 E CENTRAL,67208
683-5611
35 M 102 69 DPH
MEEKER II, 8RUCE P, 34S N HILLSIDE,67214
686-3384
30 M 1902 58 D8G
MELEAN, JAIME, 1152 SDUTH CLIFTDN,67218
681-2401
40 M 17602 65 CD
MELHORN, J MARK, 656 S LDRRAINE,67211
268-5989
53 M 1902 80 ORS
MELHDRN MD, KATHERINE J, 6S6 S LDRRAINE,67211
268-8302
S5 F 1902 81 PD
MENAKER, JERDME S, 2703 E CENTRAL,67214
685-1277
16 M 1002 41 D8G
MENDIDNES, L MARLENE, 835 N HILLSIDE,67214
685-4395
45 F 1611 70 D
MENDIONES, RUPERTD D, 3243 E MURDOCK -SUITE 404,67208
682-6585
44 M 1611 71 IM
MENEHAN, H JAMES, 3244 E DDUGLAS,67208
689-9404
26 M 1902 S3 PD
MENHUSEN, MDNTY J, D,D,, 3243 E MURDDCK SUITE 401,67208
686-7327
48 M 1676 79 ANES
MENKING, F W MANFREO, 3244 E DOUGLAS,67208
689-9336
34 M 40715 61 PD
MENKING, SUSAN MARGARET, UKSM WICHITA,67214
268-8302
41 F 3840 67 PD
MERCADER, MARID S, 2322 E CENTRAL,67214
263-6186
43 M 74801 64 ANES
MEREDITH, W TDM, 1035 N EMPDRIA,67214
263-7285
35 M 4812 61 IM
MERRITT, JDE P, 3243 E MURDDCK,67208
688-5211
46 M 2802 71 D8G

MERSHON, JAMES C, 933 N TOPEKA,67214
263-5889
37 M 1803 63 CO
MEYER, WARREN E, 1144 S CLIFTON,67218
684-5237
27 M 1606 51 GS
MICHELBACH, ALBERT P, 2916 E CENTRAL,67214
684-5243
35 M 2101 61 IM
MILFELD, DOUGLAS J, 818 N EMPORIA SUITE 200,67214
263-0296
45 M 4804 72 TS
MILLER, CLYDE W, 7618 DONEGAL,67206

09 M 2002 36 OO
MILLER, DAVID PATERSON, 7111 E 21ST N,67206
688-3070
50 M 2803 77 FP
MILLER, DON E, 4145 E KELLOGG,67218
682-6551
20 M 2802 44 GS
MILLS, CHARLES O, 1140 S WATER,67213

89 M 2002 14 OO
MILLS, GEORGE QUINTON, 550 N HILLSIDE,67214
688-2810
48 M 3006 73 PATH
MINNS, GAROLD O, UKSM-WICHITA DEPT OF MED,67214
268-8378
51 M 1902 76 IM
MIRZA, MEDD, 3333 E CENTRAL,67208
686-6683
38 M 40733 64 POS
MOHR, SANDRA NELL, 400 W CENTRAL #3105,67203

57 F 1902
MOORE, DENNIS F, 1035 N EMPORIA,67214
265-3226
36 M 2101 62 HEM
MOORE, JANE A, WESLEY MED CENTER,67214
688-2239
50 F 3901 76 EM
MORGAN, JAMES I, 3124 S SENECA,67217
522-2266
29 M 1606 53 FP
MORGAN III, LOUIS S, 8030 E KELLOGG,67207
683-3811
22 M 3901 48 FP
MORRISON, RICHARD L, 1148 S HILLSIDE,67211
684-3391
42 M 1902 67 FP
MORROW, THOMAS F, 3310 E DOUGALS,67208
685-1443
21 M 5606 46 P
MOSIER, STANLEY JAY, 3243 E MURDOCK,67208
685-8231
42 M 1902 68 FP
MUELLER, VERNETTE A, 1431 S BLUFFVIEW,67218
684-3981
17 M 2802 41 OBG
MUETH, JOAN O, 7111 E 21ST,67206
684-2858
53 F 2803 79 FP
MULLINIX, JANICE M, 3333 E CENTRAL SUITE 533,67208
687-5443
47 F 3006 73 N
MURPHY, BARRY L, 3243 EAST MURDOCK #500,67208
684-0251
45 M 1902 71 IM
MURPHY, DUANE A, 3243 E MURDOCK,67208
685-1491
32 M 1902 65 ORS
MURPHY, PAUL M, 3600 E HARRY,67218
685-1111
28 M 3006 51 R
MURPHY, PAUL W, 148 N CRESTWAY,67208

49 M 1901 82 P
MURPHY, WILLIAM R C, 818 N EMPORIA, #200, 67214
263-0296
43 M 1611 68 TS
MURRAY, KENT B, VETERANS MEDICAL CENTER,67218
263-6131
47 M 3901 73 IM
NELLIS, STEPHANIE F, 3244 E DOUGLAS,67208
689-9270
53 F 1902 79 IM
NELSON, GERALD O, 825 N EMPORIA,67214
262-3495
34 M 1902 60 PS
NELSON, RUSSELL ALAN, 3333 E CENTRAL,67208
685-5271
18 M 1902 45 PD
NELSON JR, GUST H, 3600 E HARRY,67218
685-1111
23 M 1902 46 OR
NESMITH, LESLIE W, 3333 E CENTRAL,67208
683-5611
40 M 1902 66 OPH
NEWBY, JAMES P, 818 N EMPORIA SUITE 200,67214
263-0296
34 M 1902 59 TS
NEWSOM, F CARTER, 3310 E DOUGLAS,67208
685-1443
18 M 1201 43 P
NIXON, WILLIAM A, 3333 E CENTRAL SUITE 525,67208
683-6622
16 M 1902 44 GS
NORRIS, ROBERT P, 3244 E DOUGLAS,67208
689-9232
17 M 1902 43 IM
NORTH, DORIS G, 1148 S HILLSIDE,67211
684-5257
16 F 1902 47 FP
NORTH, VICTOR, 1148 S HILLSIDE,67211
684-5257
18 M 1902 47 FP
NORTON, ROBERT K, 3244 E DOUGLAS,67208
689-9235
32 M 1001 57 PD
NOWLIN, NANCY S, 1001 N MINNEAPOLIS,67214
268-8378
47 F 1902 74 IM
NUILA, RICHARD F, 3244 E DOUGLAS,67208
689-9234
50 M 34104 76 OBG
O'DONNELL JR, LEONARD A, 8033 E DOUGLAS,67207
684-2835
27 M 1902 55 IM
O'DONNELL SR, LEONARD A, 7 ST JAMES PLACE,67206

02 M 5606 26 OO
OCHSNER, BRUCE B, 1035 N EMPORIA SUITE 235,67214
263-6273
39 M 1902 65 OPH
ODENHEIMER, BURTRAM J, 3244 E DOUGLAS,67208
689-9137
48 M 2105 73 N
OSBORNE, CONRAD C, 855 N HILLSIDE,67214
685-1381
38 M 1902 67 FP
OSIO, ANTONIO L, 1520 S CLIFTON,67218
685-2371
41 M 26404 65 EM
OSOBA, WILLIAM G, 2525 W 13TH,67203
943-9391
25 M 2802 51 FP
OUANO JR, BIBIANO B, 1431 S BLUFFVIEW STE 220,67218
684-5094
40 M 74801 63 U
OWEN, LARUE W, 810 N LORRAINE,67214
685-2207
19 M 1902 50 ANES
OWEN, PERE A, 1128 S CLIFTON,67218
684-7251
37 M 1902 64 ANES
PADGETT, WILLIAM L, 606 STRATFORD,67206

24 M 1902 51 FP
PAGE, RUTH, 1051 N STRATFORD,67206

13 F 1902 43 OO
PALMER, DAVID L, 4805 W CENTRAL,67212
945-5177
37 M 1902 63 A
PARK, ROGER WALTER, 3244 E DOUGLAS,67208
689-9217
43 M 1902 69 PD
PARKER, HAROLD L, 2932 BENJAMIN,67204
262-3765
32 M 1902 67 FP
PATTERSON, BRUCE W, 1520 SOUTH CLIFTON,67218
685-2371
46 M 1902 73 EM
PATTON, J MICHAEL, 1520 S CLIFTON,67207
685-2371
51 M 3005 78 FP
PAY, NORMAN T, ST FRANCIS HOSP,67214
268-5914
45 M 74802 68 NR
PEERY, WILLIAM H, 1001 N MINNEAPOLIS,67214
261-2650
46 M 4802 73 IM
PELLETIER JR, LAWRENCE L, UKSM-WICHITA,67214
268-8378
42 M 3501 68 IM

PENCE, CHARLES D, 3244 EAST DOUGLAS,67208
689-9468
42 M 1902 68 ORS
PENNINGTON, KATHERINE, 2113 SO BLUFF CT,67218
685-5271
16 F 1902 43 PD
PETERIE, JERRY P, 818 N EMPORIA SUITE 305,67214
264-3505
48 M 1902 75 IM
PETERS, THOMAS J, 3244 E DOUGLAS,67208
689-9190
47 M 2803 77 IM
PHAN, DUNG MY, 959 N EMPORIA S 28,67214
267-5580
48 F 94101 75 FP
PHIPPS, JACK G, 315 N HILLSIDE,67214
686-3391
21 M 1902 53 FP
PIBURN, MARVIN F, 125 N ZELTA,67206
267-4201
22 M 1803 48 GS
PICKENS, ANDREW T, WICHITA PSY PO BOX 8037,67208
684-0201
43 M 2834 69 P
PINSKER, JACOB A, 1035 N EMPORIA,67214

06 M 1902 35 IM
POLING, TERRY L, 6155 E HARRY,67218
685-2306
36 M 1902 62 FP
POLLACK, SIMON, 7523 PLAZA LANE,67206

M 36 OO
POLLAND, STEPHEN M, D.O., 1035 N EMPORIA #185,67214
265-5291
39 M 2878 68 PM
POLLOCK, ANTHONY G A, 1035 N EMPORIA STE 140,67214
264-2806
45 M 8305 71 ORS
POOLE, BERNARD T, 1035 N EMPORIA,67214
264-2806
37 M 53902 62 ORS
PORTER, GARRY L, 3243 E MURDOCK STE 400,67208
686-7351
35 M 1606 61 P
POWERS, K DEAN, 2703 E CENTRAL,67214
685-1277
23 M 1902 47 OBG
PRIETO, LUIS E, 959 N EMPORIA #203,67214
265-2613
39 M 26407 65 IM
PULLMAN, NORMAN K, 3007 E CENTRAL,67214
686-7369
21 M 3006 45 PS
PULLUM, RICHARD W, D.O., 2622 W CENTRAL,67203
945-9161
27 M 1875 57 R
PURINTON, LEW W, 1431 S BLUFFVIEW DR,67218
685-1301
23 M 1902 48 IM
PUTNAM, LYLE B, 4700 W 13TH UNIT 1-1,67212

11 M 1902 36 DO
RAGHAVAN, PARULA P, 2404 GREENLEAF CT,67226
263-6131
47 F 49501 70 IM
RAGHAVAN, PRAKASH V, 1100 N ST FRANCIS STE 240,67214
262-7662
46 M 49501 69 CD
RAMADAN, HOSSEIN, 1111 N ST FRANCIS,67214
265-2613
47 M 87501 72 CDTS
RANDALL, GEORGE R, MID-KANSAS ENT ASSOC,67211
684-2838
43 M 2802 69 OTO
RANOLES, MICHAEL J, 959 N EMPORIA,67214
265-0789
48 M 1902 73 IM
RAUSA JR, FRANCISCO C, 1148 SOUTH HILLSIDE,67211
683-4658
42 M 74808 66 IM
RAWCLIFFE JR, ROBERT A, 732 N TOPEKA,67214
267-1924
29 M 3501 55 ORS
RAZEK, ZACK A, 818 N EMPORIA SUITE 200,67214
263-0296
46 M 60501 70 CDTS
READER, G WHITNEY, PO BOX 2517,67201
263-5889
48 M 2101 70 CD
REALS, WILLIAM J, UKSM-WICHITA,67214
268-8224
20 M 3006 45 PATH

REAZIN, WALTER L, 1430 HOMESTEAD,67208
685-1381
30 M 1902 58 FP
REDDI, RAGHUNATH P, ST JOSEPH MEDICAL CTR,67218
685-1111
36 M 49521 64 RT
REED, A J, 1520 S CLIFTON,67218
685-2371
40 M 3901 65 EM
REED, D CRAMER, WESLEY MED CTR HLTH STRAT,67214
688-2080
15 M 2802 41 ADM
REED, DAVID D, 3333 E CENTRAL,67208
685-1291
43 M 1902 69 DR
REEVES, BRADFORD F, 3333 E CENTRAL,67208
685-1291
37 M 4812 62 R
REISMAN, MICHAEL ALAN, 3243 E MURDOCK SUITE 600,67208
683-5688
50 M 4804 75 DPH
RELIHAN, DONALD A, 655 N WOODLAWN,67208
684-5158
27 M 1902 54 OPH
REMPEL, JOHN H, 3333 E CENTRAL,67208
685-1812
38 M 3901 62 PS
RHOADS, JAMES P, 3244 E DOUGLAS,67208
689-9106
34 M 3520 60 IM
RHODEN, CURTIS H, 3243 E MURDOCK,67208
684-0252
33 M 1606 59 IM
RHODES, IVAN E, 3333 E CENTRAL,67208
685-1291
25 M 3901 49 R
RHODES, LOWELL M, 315 N HILLSIDE,67214
685-1461
25 M 1902 53 FP
RICHARDSON, STEWART F, 3243 E MURDOCK SUITE 400,67208
685-2321
28 M 3005 54 P
RIEDERER, ROBERT E, 1131 S CLIFTON,67218
685-4354
16 M 1902 42 FP
RIEGER, ERNEST H, 3243 E MURDOCK,67208
682-4591
29 M 1902 56 GS
RIEPE, ROGER E, WESLEY MED CENTER,67214
688-2810
46 M 1803 74 PATH
RIORDAN, HUGH D, 428 N OLIVER,67208
682-9241
32 M 5605 57 P
ROACH, NEIL E, UKSM - WICHITA,67214
268-8388
38 M 1902 67 CP
RDAN, YEAI, PERINATAL DIVISION,67214
688-2384
41 M 38501 67 PO
ROBERTS, DANIEL K, 3333 E CENTRAL SUITE 301,67208
688-3185
36 M 3005 61 OBG
ROBERTS, ROGER W, D.O., 3010 W CENTRAL,67203
945-5221
49 M 2879 75 CD
ROBERTSON, JOSEPH K, 818 N EMPORIA SUITE 200,67214
263-0296
41 M 3901 66 GS
ROBINSON, G DONALD, 3333 E CENTRAL,67208
686-6659
28 M 1902 54 PD
ROBINSON, JOHN E, 2708 E CENTRAL,67214
686-7351
32 M 6201 50 P
ROBINSON, ROBERT H, 3244 E DOUGLAS,67208
689-9445
20 M 1902 53 ANES
ROBL, DAVID A, 10300 W MAPLE,67209
722-6892
48 M 1902 74 FP
RODRIGUEZ-RAMOS, ERNEST R, 1111 N ST FRANCIS,67214
265-2613
42 M 27501 67 CDTS
RODRIGUEZTOCKER, LILIA, 1111 N ST FRANCIS,67214
265-2613
21 F 27501 49 IM
ROMALIS, BRIAN E, 3429 E DOUGLAS,67218
682-5069
39 M 6201 63 P
ROOS, MAUREEN, 1122 N TOPEKA,67214
265-2876
53 F 1902 75 FP

ROSE, SHELBY O, 3333 E CENTRAL,67208
681-2741
40 M 2012 68 PATH
ROSEN, DAVID, 818 N EMPORIA #303,67214
265-3774
48 M 1902 74 PD
ROSENBERG, THOMAS F, 2627 E CENTRAL,67214
684-0501
41 M 1642 68 A
ROSS, DENNIS LEE, 1035 N EMPORIA SUITE 105,67214
263-7285
47 M 3005 73 NEP
RUSSELL, PHILIP W, 3244 E DOUGLAS,67208
689-9351
22 M 1902 44 IM
SABIN JR, GEORGE M, 707 N MAIN,67203
268-0824
12 M 5002 39 AOM
SAOIQ, SULEMAN, 1144 N ST FRANCIS,67214
267-0159
40 M 70401 63 TS
SAEED, MOHAMMAD, 1520 S CLIFTON,67218
685-2371
42 M 70404 66 IM
SANDERS, GLORIA DEANNA, DSTEOPATHIC HOSPITAL,67203
945-9161
50 F 1902 75 PATH
SANDHU, JAGVEER SINGH, 9000 E LINCOLN #2307,67207

54 M 49611 79
SANTOSCOY, GILBERT S, 3244 E DOUGLAS,67208
689-9124
38 M 4812 62 GS
SCHILTZ, FRANCES, 115 S RUTAN #108,67218

93 F 6701 23 OO
SCHLACHTER, ERNEST R, 406 E CENTRAL,67202
265-0705
24 M 1902 52 FP
SCHLICHER, JOHN E, 3244 E DOUGLAS,67208
689-9346
40 M 1803 66 D
SCHLUETER, JOHN J, 3333 E CENTRAL,67208
685-9289
31 M 3841 56 R
SCHNELLE, JOACHIM, 4145 EAST KELLOGG,67218
682-0621
44 M 40933 70 FP
SCHOPF, CLIFTON C, 222 S RIDGE RD,67209
945-0142
29 M 1902 57 FP
SCHWARTZ, V DEAN, 400 N WOODLAWN #4,67208
684-3881
24 M 1902 48 FP
SCOTT, VINCENT L, 641 N WOODLAWN #43,67208

03 M 3806 29 OO
SCOTT, WILLIAM H, 1431 S BLUFFVIEW STE 111,67218
685-1111
41 M 4901 65 CO
SEN SARMA, PRONAB K, 1144 N ST FRANCIS,67214
267-0159
45 M 49518 71 CO
SHAFER, PRESTON J, 3333 E CENTRAL,67208
685-7234
20 M 3005 46 OBG
SHAH, MUKHTAR H, 3243 E MUROOCK STE 400,67208
686-2994
40 M 70404 63 P
SHAW, RICHARD C, 825 N EMPORIA,67214
262-3495
35 M 1902 61 PS
SHEHI, LORA J, 834 CARTER,67203

56 F 1902
SHELLITO, JOHN G, 3244 E DOUGLAS,67208
689-9124
18 M 1606 43 TS
SHIELD, CHARLES, 818 N EMPORIA SUITE 200,67214
263-0296
46 M 2802 72 GS
SHOFFNER, RICHARD W, 3244 E DOUGLAS,67208
689-9271
53 M 3979 79 IM
SHRADER, C ERIC, 655 N WOODLAWN,67208

47 M 1902 78 OPH
SHRADER, ODYLE A, 3333 E CENTRAL,67208
682-4851
16 M 1902 41 EENT
SHURTZ, GLEN L, 3333 E CENTRAL SUITE 214,67208
685-1291
40 M 4802 78 R

SIEGEL, ALBERT R, 3600 E HARRY,67218
685-1111
22 M 1642 47 PM
SIFFORD, R LAWRENCE, 959 N EMPORIA,67214
265-0561
25 M 1803 52 IM
SKIBBA, RICHARD M, 3244 E DOUGLAS,67208
689-9477
43 M 5606 70 GE
SLUTSKY, LAWRENCE JOEL, ST FRANCIS HOSPITAL,67214
268-5922
46 M 3501 72 OR
SMITH, ALVIN L, 929 N ST FRANCIS,67214
262-6211
28 M 5606 57 PATH
SMITH, TIMOTHY WM, 1035 N EMPORIA STE 270,67214
269-4026
49 M 1902 74 IM
SMITH JR, WILLARD J, 851 N HILLSIDE,67214
685-1371
32 M 1611 57 U
SNYDER, GREGG M, 902 N HILLSIDE,67214
685-2377
27 M 1803 54 NS
SOLOMON, HERMAN, 835 N HILLSIDE,67214
685-4395
37 M 2701 62 O
SOLTZ, ROBERT A, 3244 EAST DOUGLAS,67208
689-9381
47 M 2803 74 PD
SOMERS, MARVIN M, ST FRANCIS HOSP,67214
262-6211
23 M 1902 48 R
SPANN, RICHARD W, 3243 E MUROOCK,67208
684-0252
40 M 1902 65 PUD
STAMPS, PHIL, ST JOSEPH MEDICAL CENTER,67218
685-1111
37 M 3901 63 PATH
STANLEY, KENNETH E, 959 N EMPORIA,67214
267-0256
31 M 1902 56 U
STARK, JAMES R, 3244 E DOUGLAS,67208
689-9422
20 M 1902 44 R
STECKLEY, RICHARD ALLEN, 1035 EMPORIA SUITE 130,67214
263-3271
49 M 2105 74 IM
STEIN, PAUL S, 3243 E MUROOCK,67208
685-2377
40 M 3305 66 NS
STEMBRIDGE, TRAVIS W, 3333 E CENTRAL SUITE 301,67208
685-7234
47 M 4802 76 ORG
STOLZ, ELMER G, 2349 MC LEAN BLVD NW,67204
265-6287
17 M 3006 45 FP
STREET, DAVID E, 818 N EMPORIA SUITE 200,67214
263-0296
35 M 2101 61 GS
STREIT, JEROME G, 1131 S CLIFTON,67218
685-4354
48 M 1902 77 FP
STRICKLAND, MAURICE VAN, 3244 E DOUGLAS,67208
689-9156
51 M 4804 74 A
STRYKER, TERRY MARGARET, 3243 E MUROOCK - STE 300,67208
685-8231
47 F 1002 75 FP
SUERD, JESUS T, 1148 S HILLSIDE,67211
681-3371
33 M 74802 57 PUD
SULLIVAN, CORNELIUS J P, 1431 BLUFFVIEW DR,67218
685-4851
18 M 3509 43 OBG
SULLIVAN, LEONARD L, 3244 E DOUGLAS,67208
689-9454
35 M 1902 61 PD
SVOBODA, LOIS V, 3243 E MUROOCK STE 300,67208
685-8231
39 F 1602 66 FP
SVOBODA, WILLIAM B, 3243 MURDOCK SUITE 601,67208
685-2377
36 M 1602 63 PDN
SWEET, DONNA E, UKSM-WICHITA,67214
268-8378
48 F 1902 79 IM
TATPATI, DANIEL A, 1144 N ST FRANCIS,67214
267-0159
44 M 49535 67 TS

TATPATI, OLGA ADELINA, U K S M - WICHITA,67214
268-5992
44 F 49535 67 PD
TAYLOR, RICHARD J, 929 N ST FRANCIS,67214
262-1952
21 M 3006 49 PATH
TAYLOR, STEVEN L, USAF HOSP,67221
681-5866
46 M 1902 77 GP
THELEN, J CHRISTINE, 1738 N ROOSEVELT,67208
-
13 F 5104 37 OO
THOMPSON, DANIEL M, PO BOX 4044,67204
838-3381
19 M 1902 50 FP
THOMPSON, SCOTT W, 1632 W 22ND #407,67204
838-0994
51 M 64914 78 FP
TIHEN, EDWARD N, 3244 E DOUGLAS,67208
689-9481
24 M 1606 48 IM
TIHEN, HENRY N, 1227 N RIVER BLVD,67203
-
96 M 1601 19 OO
TILLER, GEORGE R, 5101 E KELLOGG,67218
684-5255
41 M 1902 67 AM
TILTON, FRANK M, 3244 E DOUGLAS,67208
689-9137
33 M 2002 59 N
TINKER, ROBERT C, 1126 S CLIFTON,67218
682-7578
31 M 2834 57 ORS
TINTEROW, MAURICE M, 3333 E CENTRAL,67208
685-4389
17 M 4802 41 ANES
TIPPIN JR, ERNEST E, 959 N EMPORIA,67214
265-5256
24 M 1902 50 OTO
TOCKER, ALFRED M, 1111 N ST FRANICS,67214
265-2613
15 M 4802 40 COTS
TONN, GERHART R, 855 N HILLSIDE,67214
685-1381
16 M 1902 44 FP
TOOHEY, JOHN S, 3244 E DOUGLAS,67208
689-9175
50 M 5605 77 ORS
TOSH, FRED E, 1900 E NINTH,67214
268-8391
30 M 4706 54 PH
TRACY, TERRY A, 3333 E CENTRAL,67208
685-7234
35 M 2803 61 OBG
TRAVERS, HENRY, WESLEY MED CTR LAB MED,67214
688-2812
46 M 4114 71 PATH
TRETBAR, HARVEY A, 3243 E MURDOCK #500,67208
684-0251
25 M 1902 52 IM
TREWEEKE, MICHAEL W, 2916 EAST CENTRAL,67214
684-5234
46 M 1902 72 IM
TRUDEAU, DAVID L, ALCOHOL TREATMENT UNIT,67218
685-1111
40 M 2604 66 AOT
TRUJILLO, ANTERO A, 3600 E HARRY,67218
685-1111
36 M 73701 61 ANES
UHLIG, PAUL J, 3244 E DOUGLAS,67208
689-9191
28 M 1902 57 PD
ULRICH, BRIAN K, 1035 N EMPORIA SUITE 265,67214
265-3226
48 M 1902 74 IM
VAN LEEUWEN, GERALD, UKSM-WICHITA,67214
268-8221
29 M 1803 54 NEO
VIN ZANT, LARRY E, 1431 BLUFFVIEW STE 109,67218
682-6532
10 M 1902 40 GS
VINE, DONALD LEE, 3244 E DOUGLAS,67208
689-9240
39 M 511 66 CO
VINZANT, WHITNEY L, 1431 SOUTH BLUFFVIEW #109,67218
686-1991
45 M 1902 71 GS
VLIET, LEE W, UKSM WICHITA,67214
268-8388
46 F 5107 78 P
VORHEES, VICTOR J, 3243 E MURDOCK,67208
685-8231
36 M 1902 68 FP

VOTH, DOUGLAS W, UKSM-WICHITA DEPT OF IM,67214
268-8378
34 M 1902 59 IM
WAOUD, ABOUL, 1543 S HILLSIDE,67211
682-6814
35 M 70409 60 P
WALTERS, ROBERT MERRILL, 3244 E DOUGLAS,67208
789-9495
41 M 1601 74 ORS
WARREN, LLOYD P, 5205 E 21ST,67208
683-7223
11 M 1902 36 OPH
WARREN, WIRT A, 2226 S MINNESOTA,67201
267-0520
09 M 2802 33 PGER
WARREN JR, JOHN W, 931 N YALE,67208
-
15 M 2501 39 OO
WEAVER, J ROBERT, 959 N EMPORIA,67214
265-5731
21 M 1902 48 FP
WEAVER, JACK O, 959 N EMPORIA,67214
265-7241
16 M 2802 42 OPH
WEBER JR, HUGO P, 1035 N EMPORIA,67214
263-7285
40 M 702 66 IM
WEBSTER, BOBBY W, 550 N LORRAINE,67214
685-0559
48 M 4802 74 OBG
WEDEL, A K, 621 S ROOSEVELT,67218
-
56 M 1902 82 FP
WEIPPERT, EDWARD J, 10300 W MAPLE,67209
722-6892
44 M 1902 70 FP
WELCH, ANNA L, 130 N SEDGWICK,67203
-
48 F 1720 81 FP
WELCH, LAUREN K, 3243 E MURDOCK SUITE 601,67208
685-2377
35 M 1902 61 N
WELCH, MARTIN H, UKSM-WICHITA,67214
268-8378
36 M 5605 61 IM
WELLS, MAX MICHAEL, 550 N HILLSIDE,67214
688-2820
50 M 1902 76 PATH
WELLSHEAR, CHARLES C, WICHITA PSY CENTER,67208
684-0201
30 M 4706 58 P
WENINGER, JOHN H, 1148 S HILLSIDE,67211
682-6523
32 M 3005 62 FP
WEST, WILLIAM T, 3244 E DOUGLAS,67208
689-9234
24 M 1902 49 OBG
WHALDON, JACOB T, 202 N ROCK RD APT 120,67206
-
14 M 1720 41 OO
WHEELER, NICKY RAY, 3007 E CENTRAL,67214
686-7369
48 M 1902 74 PS
WHEELER, PINCKNEY R, 2208 W 13TH,67203
943-2118
18 M 3901 56 FP
WHITAKER, JAMES A, 3243 E MURDOCK,67208
684-0251
44 M 1902 72 IM
WHITE, CHARLES M, 3244 E DOUGLAS,67208
689-9422
15 M 3005 41 R
WILDER, LOWELL W, 655 N WOODLAWN,67208
684-5158
35 M 4109 62 OPH
WILKINSON, LARRY K, 1520 S CLIFTON,67218
685-2371
46 M 1902 74 FP
WILLIAMS, BRUCE N, 1122 N TOPEKA, 67214
268-5996
54 M 4002 80 FP
WILLIAMS, CHARLES L, 3244 E DOUGLAS,67208
689-9268
16 M 2834 43 IM
WILLIAMS, HOWARD V, 1439 GOEBEL CIRCLE,67207
268-8251
18 M 1205 43 P
WILLIAMSON, STEPHEN K, VA MEDICAL CTR, 67218
685-2221
54 M 1902 79 IM
WINCHELL, H H FORSYTH, PO BOX 2579,67201
265-1684
34 M 3545 58 R

WINCHESTER, EUGENE B, 2601 E CENTRAL,67214
684-0271
18 M 3006 56 FP
WISDOM, JAY K, 1148 S HILLSIDE,67211
684-0271
12 M 1902 42 FP
WISNER JR, HARRY J, 3244 E DOUGLAS,67208
689-9169
17 M 3005 43 1M
WITTMANN, ALBERT F, 2323 N WOODLAWN,67220
-
10 M 2834 38 OD
WOLFE, FREDERICK, 6611 E CENTRAL,67206
686-1152
36 M 3508 66 RHU
WOOD, GARY A, 2450 LONGWOOD CIRCLE,67206
-
52 M 64936 81
WOOD, GARY B, 3243 E MURDOCK #404,67208
684-2131
21 M 2802 45 1M
WOOD, ROBERT D, 2337 RUTLAND CT,67226
486-2127
26 M 1902 53 FP
WOODHOUSE, CHARLES L, 959 N EMPORIA,67214
265-8821
10 M 1902 34 ENT
WOODRING, CATHY S, 222 S RIDGE RD,67209
945-0142
51 F 3546 77 FP
WORSING JR, ROBERT A, 3244 E DOUGLAS,67208
689-9175
47 M 2604 72 ORS
WRAY, ALEXANDER J, 120 E 21ST ST,67214
838-4912
19 M 1902 49 FP
WRENN, C J, 3244 E DOUGLAS,67208
689-9156
47 M 3005 73 A
WU, JIN-TZE, 3333 E CENTRAL SUITE 214,67208
688-2920
41 M 38502 67 TR
YOCKEY, CHARLES C, 3243 E MURDOCK S 500,67208
684-2051
46 M 1902 72 IM
YOON, CHANG SUP, 3243 E MURDOCK SUITE 401,67208
686-7327
46 M 58303 72 ANES
YOUNG, DOUGLAS L, 3244 E DOUGLAS,67208
689-9213
42 M 1902 71 1M
YOUNG, ROBERT, 3243 E MURDOCK,67208
688-5211
48 M 1902 78 OBG
YOUNGBERG, DEAN 1, 2916 EAST CENTRAL,67214
684-5243
46 M 1902 72 1M
ZARNOW, HILARY, 1624 GATEWOOD,67203
263-3219
45 M 1611 69 R
ZATZKIN, JAY B, 1035 N EMPORIA,67214
262-4467
46 M 2002 74 ONC
ZEPICK, LYLE F, 933 N TOPEKA,67214
263-5889
50 M 6001 71 CO
ZIMMERMAN, KENNETH D, 934 CRESTLINE,67212
687-3925
29 M 3901 55 OM
ZONGKER, PHILIP E, 3244 E DOUGLAS,67208
689-9422
43 M 1902 70 R

WINCHESTER—913
(Jefferson County)

HUSTON, FRANCIS W, ,66097
-
06 M 1601 34 FP

WINFIELD—316
(Cowley County Society)

CARRO, F AURELIO, PO BOX 544,67156
221-3200
23 M 27501 49 1M
DAEHNKE, SIGURD S, PO BOX 544,67156
221-3200
26 M 3005 60 FP
FOWLER, DENNIS L, PO BOX 544,67156
221-2300
48 M 1902 73 ES
HUTCHINSON, DIRK T, PO BOX 544,67156
221-3200
48 M 3901 74 1M
KAUFMAN, LELAND R, 221 WEST 8TH,67156
221-3350
33 M 1902 61 FP
KAUL, ANAND N, PO BOX 544,67156
221-3200
39 M 49530 61 1M
LAWRY, JAMES VORIS, 1017 E EIGHT,67156
221-3200
40 M 502 77 GP
MAC KILLOP JR, DANIEL, 4 FLEETWOOD DR,67156
-
11 M 2407 38 OD
MILLER, FRANKLIN R, BOX 544,67156
-
02 M 2401 27 OD
NARAWONG, DUANGCHAI, PO BOX 544,67156
221-3200
49 F 89104 74 PD
NEMMERS, DAVID J, PO BOX 544,67156
221-3200
34 M 1803 59 1M
SAMUEL, CHANDY C, 1211 E 5TH,67156
221-6100
35 M 49527 59 GS
SAMUEL, SHANTHI, 5 FLEETWOOD DR,67156
221-2300
42 F 49527 67 PATH
SHAH, ASHOK H, PO BOX 544,67156
221-3200
41 M 49548 66 OBG
SNYDER, C JOHN, 1317 WHEAT RD,67156
221-3200
38 M 2401 64 GS
SNYDER, HOWARD E, 1317 WHEAT ROAD,67156
-
03 M 4102 27 OD
WELLS, ALVIN Y, 1707 E 10TH,67156
-
97 M 1902 37 OD
WELLS, BRUCE W, 221 W 8TH ST,67156
221-3350
39 M 1902 64 1M
WHITE, R BURNLEY, 117 W 9TH,67156
221-2950
24 M 1902 52 FP
WINBLAD, J KENT, 15 FLEETWOOD,67156
221-6100
51 M 1902 76 OBG
WINBLAD, JAMES N, 1211 E 5TH ST,67156
221-6100
27 M 1902 53 GS

YATES CENTER—316
(Allen County Society)

ATKIN, JOHN D, 1004 E MADISON,66783
625-2312
35 M 3901 61 FP

OUT OF STATE

ALLEN, MARK LYNN, 3720 WYOMING SUITE 3
KANSAS CITY MO 64111
ANDERSON, LYLE B, 4745 S STATE RD #446
BLOOMINGTON IN 47401
ANDERSON, WILLIAM A, 629 WEST 70TH
KANSAS CITY MO 64113
AUCHARD, VIRGIL M, 1170 W MANSFIELD
BUCYRUS OH 44820
BACON, ARTHUR H, 38 RUBBER TREE DR
LAKE WORTH FL 33643
BARNES, MARIAN, 2040 W AVE J-13 APT 27
LANCASTER CA 93534
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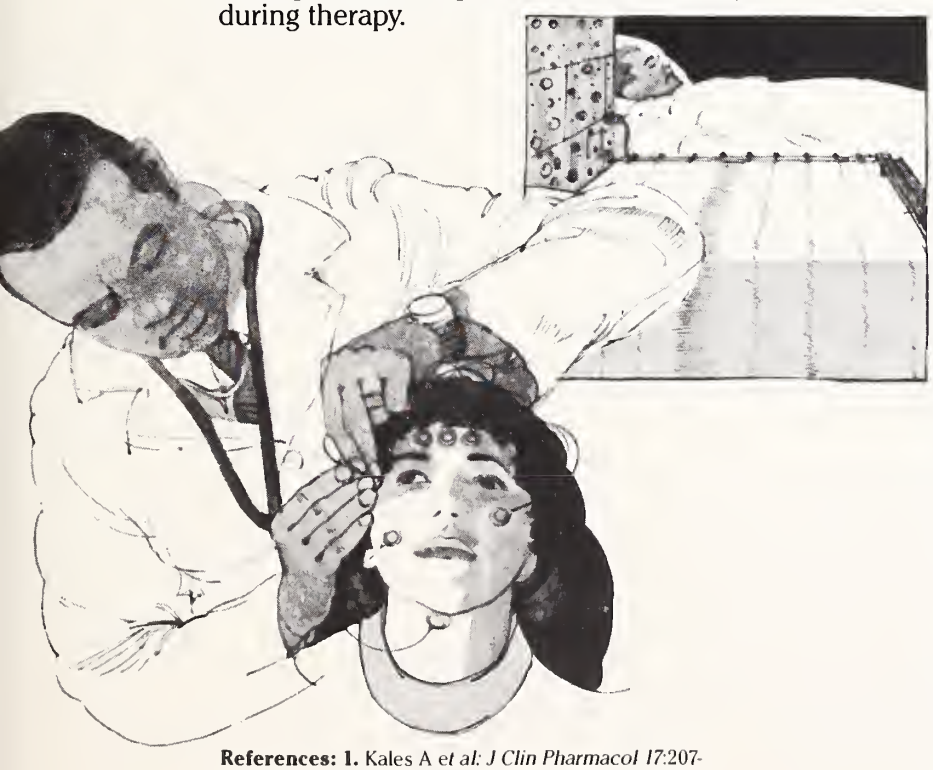
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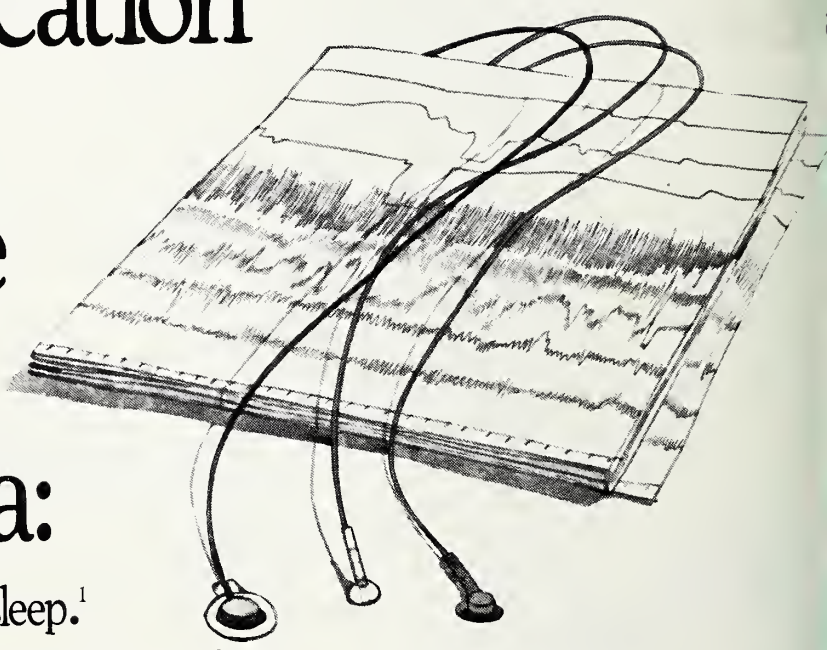
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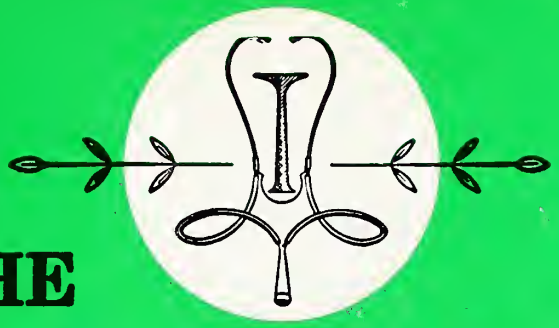
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Council Meeting

Report of Meeting Held July 30, 1983

A meeting of the Council was convened by Jimmie A. Gleason, M.D., President, on Saturday, July 30, 1983, at the Salina Hilton Inn, beginning at 10:00 A.M. Present were Drs.: F. Calvin Bigler, James G. Bridgens, K. William Bruner, Jr., Clair C. Conard, Louis M. Culp, Herbert D. Doubek, Herbert Fransen, Jimmie A. Gleason, Roman Hiszczynskyj, William T. King, David A. Leitch, Wendale E. McAllaster, Warren E. Meyer, John R. Neuenschwander, Lew W. Purinton, Edwin D. Rathbun, Alex Scott, Newton C. Smith, Wayne O. Wallace, Jr., Linda D. Warren, Wallace N. Weber, Kermit G. Wedel, and Emerson D. Yoder. Staff present were Steve Carter, Gary Caruthers, and Jerry Slaughter.

The Council approved the following actions of the Executive Committee:

- Refund of half-year dues to H. Tom Gray, M.D., Wichita, due to retirement; and E. Fred Elledge, M.D., Great Bend, due to leaving Kansas;
- Reassignment of former Edwards County Medical Society members to the surrounding neighboring county medical societies;
- A $250 contribution to the Social Security Disability Workshop;
- A $250 contribution to the Kansas Silverhaired Legislature;
- Accepted the Riley County Medical Society invitation to hold the 1987 Annual Meeting in Manhattan.

The Council considered the proposal to hold the 1988 Annual Meeting in Kansas City. The Councilors from Wyandotte and Johnson counties will discuss the possibility of staging the convention on the Plaza in Kansas City, Missouri, and will report back to the Council.

The 1984 Nominating Committee was selected as follows:

Kermit G. Wedel, M.D., Minneapolis, Chairman
David M. Pugh, M.D., Kansas City
James H. Ransom, M.D., Topeka
Edwin D. Rathbun, M.D., Liberal

The request by Cherokee County Medical Society to combine with the Crawford County Medical Society was approved.

Dr. Wedel, member of the HCFA Physician Advisory Committee, reported on the first organizational meeting of the Health Care Financing Administration, under the Department of Health and Human Services. The primary purpose of the meeting was to identify the purpose of the committee, to study the issues of physician reimbursement, and to prepare a report for the 1985 Congress.

The Council heard a report by Mr. Slaughter concerning the professional liability situation in Kansas. The Health Care Stabilization Fund currently has accrued liability of $26 million. The study of the financial position of the Fund was done by an independent actuary. The KMS Professional Liability Committee has been studying this matter and recommends the following options for Council consideration:

1. Remove the statutory $10 million maximum limit on the Health Care Stabilization Fund.
2. Remove the statutory maximum 65 per cent surcharge and allow the surcharge to increase as needed to ensure the Health Care Stabilization Fund financial solvency on an accrual accounting basis over a five-year period.
3. Increase the basic insurance coverage reimbursement from $100,000/300,000 to $200,000/600,000, if coverage availability can be assured.
4. Retain the unlimited coverage feature of the Health Care Stabilization Fund.
5. Work with the Insurance Commissioner to develop a non-legislative advisory panel of physicians and other health care providers to assist in claims review and management of the Fund.

The Council approved recommendations 1, 2, 3, and 5. Mr. Slaughter clarified that on point 3, the Council was endorsing the concept of periodically indexing the basic coverage as needed, including the proposed figures raising the basic coverage limit to $200,000/600,000 for the years 1984, 1985 and 1986, and raising it to $250,000/750,000 in the years 1987, 1988 and 1989.

In discussing point 4, the Council voted to retain the unlimited coverage feature of the Health Care Stabilization Fund with the option of the sliding scale which would allow physicians to elect the level of coverage they wanted on an annual basis.

The Council then instructed the Professional Liability Committee to develop a plan for educating KMS members on prevention of professional liability actions.

The discussion of in-hospital peer review was referred for consideration at the next meeting of the Council.

There being no further business, the meeting adjourned at 12:15 P.M.

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AUXILIARY NEWS

An Open Letter to Kansas Physicians

The Kansas Medical Society Auxiliary held the Forum on Aging and the Summer Board Meeting at the Holidome in Lawrence, July 13-14, 1983. Shirley Reese (John) was the capable and efficient meeting chairman, graciously assisted by Jo Price (Laurance), Douglas County Auxiliary President. Polly Reed (James), candid camera person, and Shirley Wilcox (Howard), chronicler of the proceedings, are just two of the many Douglas County Auxiliary members who helped.

The Medical Society staff did a magnificient job of coordinating the Forum on Aging, which was held on the 13th. Besides the Medical Auxiliary with Harriett Olson (Carl) and Ruth Mitchell (Alex) from Lawrence serving as state co-chairmen on Aging, the other co-sponsoring organizations were: The Kansas Department on Aging, Kansas Insurance Department, Kansas Department of Health and Environment, Medicare, and the Kansas Medical Society.

The forum commenced with a luncheon speech entitled "Alzheimer's Disease, A Community and Family Issue," by Linda Wright, R.N., head of Inservices, Clinicare Family Health Services, Kansas City. This particular speech, as well as the luncheon, will not be repeated at each county forum, but we found the subject to be very interesting.

The topic of Medicare was broken down as follows: "Present Coverage," discussion by Miriam Clark, Supervisor of Medicare Training; "Supplemental Coverage," discussion by Waynette Detter, Consumer Assistance Division, Kansas Department of Insurance; and "Future Changes," discussion by L. Jay Lohmann, Kansas Director of Medicare.

Three speakers dealt with long term care: Gary Petz, Director of Planning, Kansas Department on Aging; Mary Corrigan, Acting Director, Office of Health Planning, Kansas Department of Health and Environment; and Julie Rosenzweig, Douglas County Health Department.

"How to Ask Your Physician" was the subject of the talk by Alex Scott, M.D., Chairman, KMS Committee on Aging.

For the finale, Senator Jan Meyers, Chairman of the Senate Public Health and Welfare Committee, gave a legislative overview.

As you can judge from the subjects and speakers, this was a dynamic, excellent forum. This program was to serve only as a model for the county auxiliaries to use in their communities. The Kansas Medical Society executive office will organize the program for the county forums. The local auxiliaries will be responsible for the mechanics of locating a meeting place, refreshments, and drawing a crowd. Please encourage your local auxiliary to participate. This is a wonderful and worthwhile opportunity to inform the community and to do needed public relations work at the same time.

On July 14, the State Auxiliary held its board meeting. Nancy Roderick (James), Salina, State Drug and Alcohol Abuse chairman, solicited the Auxiliary's support to help coordinate state activities for the PBS television program, "Chemical People," November 2 and 9, 8:00 P.M. (eastern time), to be hosted by First Lady Nancy Reagan. This is a project deserving the support of the medical community.

Wanda Macy (Norman) and Ruby Reece (Richard), Salina, state co-chairmen for child abuse, presented a videotape program entitled "The Bubbylonian Encounter." Wanda and Ruby will bring

this excellent program on prevention of sexual abuse of children to your community, if requested.

Dot Meyer (Warren), a member of the KaMPAC Board, gave an impassioned plea for membership. The message "hit home" and we had an excellent recruitment program on the spot. Medicine's only hope for survival is to take an active stand; KaMPAC affords this opportunity. Please, Kansas physicians, join up and also include your spouse's membership.

In conclusion, this was a very stimulating but somewhat unsettling two-day meeting. Medicine is at the crossroads in many areas, and "Now is the Hour" for the Auxiliary to participate actively and to assist in helping Medicine stay alive and well.

Diane Sanders
President
Kansas Medical Society Auxiliary

PATIENTS NEEDED
at Kansas University Medical Center

- Headache patients: age 18-60, for comparative therapy study sponsored by the National Institutes of Health. Must have average 2-20 episodes per month & be willing to come to Headache Clinic once monthly for tests (3 hrs each time). All studies and medications free.
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- Patients with multi infarct dementia for a cooperative clinical trial of a new and promising drug therapy.

Contact Dept. of Neurology, 1-913-588-6970.

ARTHRITIS SYMPOSIUM

DATE: Wednesday, October 19, 1983 **TIME:** 1:00-5:00 P.M. 6:30-8:30 P.M.

PLACE: St. Francis Regional Medical Center
Educational Center Auditorium
929 N. St. Francis
Wichita, Kansas

Course Credit: Course is approved for 5 hours — Category 1, CME Credit; Category D2 has been applied for.

FEE: $10.00

FACULTY

Emmett Lunceford, M.D., Professor of Orthopedic Surgery, University of South Carolina
Joseph J. Weiss, M.D., Formerly Assistant Professor of Medicine, University of Michigan
Roy D. Altman, Professor of Medicine, University of Miami School of Medicine
Richard Evans, M.D., Assistant Professor of Medicine, University of Kansas — Wichita

Moderator: Frederick Wolfe, M.D., Symposium Chairman

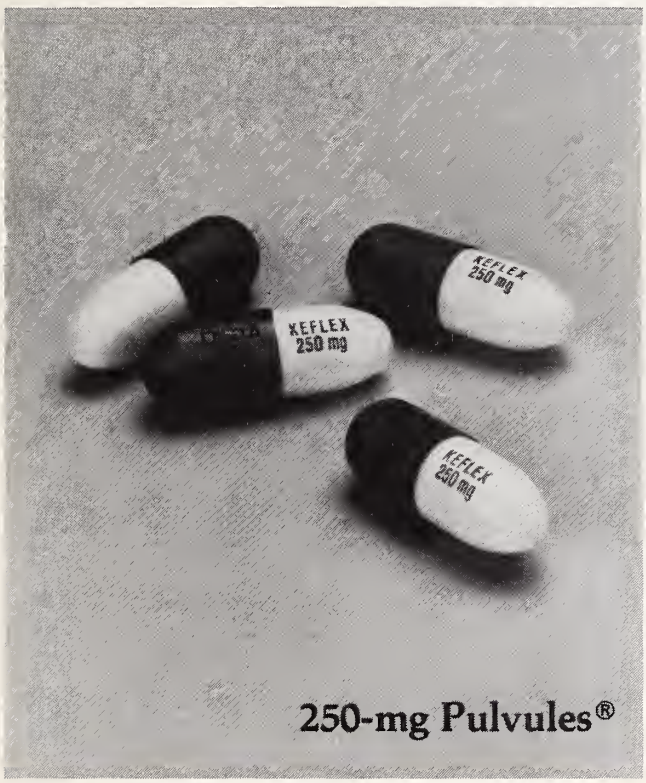
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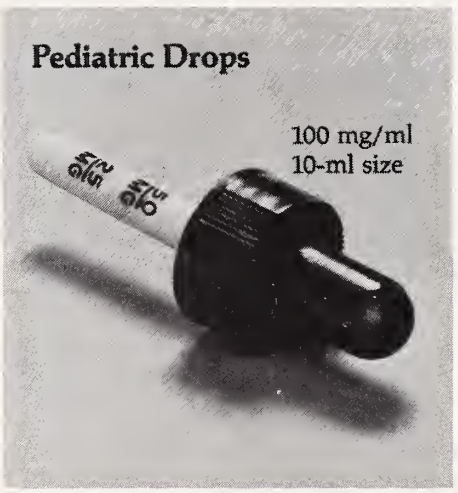
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AMA House of Delegates

Summary of Actions Taken at the 1983 Annual Meeting, Chicago

• The AMA House of Delegates met in Chicago June 19-23.

• There were 351 delegates seated:

— 281 delegates representing state medical associations;
— 61 delegates representing national medical specialty societies;
— 9 Section and Service delegates representing hospital medical staffs, medical students, medical schools, resident physicians, Army, Navy, Air Force, USPHS, and the Veterans Administration.

• The American Hospital Association and the American Dental Association also sent official observers. At this meeting the House voted to also invite the American Osteopathic Association, American Group Practice Association, and the National Medical Association to send official observers.

• The President of the United States spoke to the House of Delegates on Thursday, June 23. Topics included:

— Support for federal research for medicine and health;
— Support for the Orphan Drug Act and his hope that the legislation will provide incentives for the private sector to develop drugs to treat rare diseases;
— The cost of health care and proposals for a one-year freeze on Medicare physician reimbursement and co-payments for Medicaid.

The President began his remarks by saying "the quality of American medicine is unsurpassed and on that we don't need a second opinion. . . . My respect for your profession is deep and personal."

The President congratulated the AMA on its cost effectiveness programs and its Health Policy Agenda. He also thanked those medical societies that have private sector programs for the needy and unemployed.

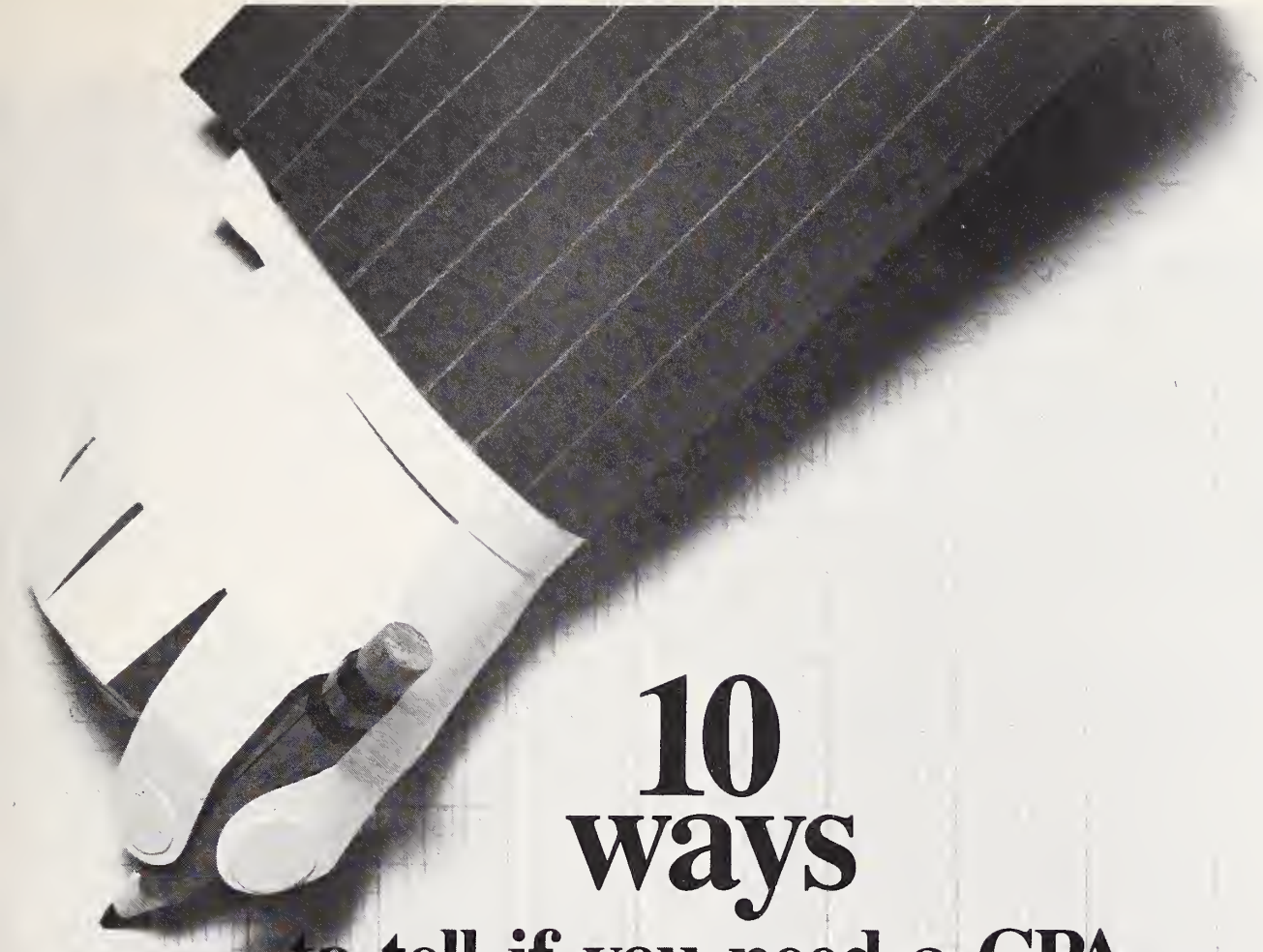
• Hospital Medical Staff Section: Nearly 700 representatives of hospital medical staffs met for the first time in conjunction with the AMA House. Section Officers were elected including an AMA delegate. The Section introduced eight resolutions. There was great enthusiasm for bringing hospital medical staffs into the AMA policy-making apparatus and giving medical staffs an important voice on issues affecting them. With 7,000 U. S. hospitals eligible to send representatives, attendance is expected to grow. The Section will meet again at the Interim Meeting in Los Angeles and *all hospitals are urged to send representatives*. The Section will meet December 2-4 and the AMA House meets December 4-7.

• The House considered 147 resolutions and 74 reports.

JCAH Accreditation Manual for Hospitals

After extensive debate on the Medical Staff Section of the JCAH *Accreditation Manual for Hospitals,* the House commended the Board of Trustees for its responsible consideration of these difficult issues. The House also affirmed the following principles as the basis for any revisions in the JCAH Manual:

- Continue the use of the term "Medical Staff" in the title of the chapter and throughout the Manual.
- Delete any specific references to limited licensed practitioners now contained in the Medical Staff chapter of the 1983 *Accreditation Manual for Hospitals*. This does not preclude such practitioners from having hsopital privileges consonant with their training, experience, and current competence if approved by the normal credentialing process.
- Provide consideration of qualified limited licensed practitioners in accordance with state law and when approved by the executive committee of the medical staff and by the governing board and, when their services are appropriate to the goals and missions of that hospital, taking into account the training, experience, and current clinical competence of the practitioners.
- Provide that the executive committee of the medical staff is composed of members selected by the medical staff, or appointed in accordance with the hospital bylaws. All members of the active medical staff, as defined in the Medical Staff Bylaws, are eligible for membership on the executive committee, and a majority of the executive committee members must be fully licensed physician members (Doctors of Medicine or Doctors of Osteopathy) of the active medical staff in the hospital.
- Assure that the medical care of all patients re-



10 ways to tell if you need a CPA.

- ☐ **1** Are your tax returns getting complicated? (Perhaps because family earnings are now from *several* sources or because your business is growing or changing?)
- ☐ **2** Do you need *immediate* tax write-offs through proper tax planning?
- ☐ **3** Would you consider changing some business procedures to cut *future* taxes?
- ☐ **4** Are you setting up or changing estate plans for yourself or someone else?
- ☐ **5** Is your business required to file financial reports — anything from a statement of earnings to full-scale audited financial statements?
- ☐ **6** Do you feel it might be time to incorporate your business?
- ☐ **7** Are you preparing a business loan application — and need supporting documentation?
- ☐ **8** Do you want to set up or improve a bookkeeping system?
- ☐ **9** Are you thinking of starting a pension or profit-sharing plan?
- ☐ **10** Do you need an *independent* source of professional management advice on how to use your resources more efficiently or how to make financial projections?

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KS CPA

Kansas Society of Certified Public Accountants

mains under the supervision and direction of qualified, fully licensed physicians (Doctors of Medicine or Doctors of Osteopathy).
- Assure that the continued high quality of care, credentialing of physicians and other licensed practitioners, and effective quality assurance programs remain under the supervision and direction of fully licensed physicians.

Payment for Physicians' Services

Another issue that generated lengthy discussion was a report presenting an analysis of payment mechanisms utilized by third party payors for reimbursement of physicians' services.

The Council on Medical Service presented a comprehensive discussion of *indemnity vs UCR reimbursement* and asked the delegates to consider the matter and discuss it with their constituents during the next six months in preparation for discussion and possible action at the 1983 Interim Meeting in Los Angeles.

The Council believes that if third parties change to an indemnity system of payment, patients would be benefitted by:

- Insuring their continued access to care not through external regulation of fees but through market forces.
- Increasing both physicians' and patients' sensitivity to costs and quality of care provided.
- Allowing them continued freedom of choice rather than being increasingly restricted to "participating" providers as a condition of coverage.
- Facilitating understanding and comparison of insurance coverages.

The report states that rate determination for third parties would be simpler.

For physicians, the Council believes that the indemnity approach could bring improved patient/physician interaction by eliminating false expectations of the amount of the third party payment. This approach, the report states, will also provide physicians the freedom to charge what they believe to be a fair and equitable fee, subject only to normal and effective market constraints.

Resident Board Member

The House voted to provide for a position on the Board for a resident physician:

- Elected by the House for a two-year term;
- Maximum of three terms;
- Constitution and Bylaws must be changed at the Interim Meeting;
- Election would be held at the 1984 Annual Meeting. Nominations may be submitted by any delegate.

Medical Student Nonvoting Board Member

The House also voted to create a nonvoting medical student position on the Board:

- By annual appointment by the Board from two or more nominations presented by AMA Medical Student Section Governing Council;
- Individual eligible for reappointment as long as he or she remains a medical student member of the AMA;
- Constitution and Bylaws must be amended at the Interim Meeting.

Cost Shifting

The House adopted a resolution calling upon the Association to conduct a study of the effects of cost shifting on physicians, third party payors, and hospitals.

- The resolution indicated that there is "growing evidence that inadequate reimbursement by government funded third party programs has forced hospitals to shift more costs to patients enrolled in private health insurance plans."
- In addition the resolution says that proposals such as prospective reimbursement and vouchers for Medicare have the potential to exacerbate the cost shifting problem.

TEFRA Regulations

After modifying a Louisiana Resolution pertaining to TEFRA regulations, the House voted to:

- Continue to give a high priority to prompt withdrawal of regulations to implement Section 108 of TEFRA relating to the Medicare program's payment of physician services.
- The AMA was successful in delaying implementation of the regulations and in urging withdrawal.

Definition of "Physician" (Medicare)

The House modified its existing definition of "Physician" to include Doctors of Osteopathy. The definition now reads:

A physician is a person who, having been regularly admitted to a medical school or a school of osteopathic medicine duly recognized in the country in which it is located, has successfully completed the prescribed course of studies in medicine or osteopathic medicine and has acquired the requisite qualifications to be legally licensed to practice medicine or osteopathic medicine.

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for (lorazepam) ℞

Anxiety

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NEW TABLET DESIGN 0.5, 1.0 and 2.0 mg

Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® (IV) for (lorazepam) Anxiety

DOSAGE: Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.

HOW SUPPLIED: 0.5, 1.0 and 2.0mg tablets.

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In addition the House voted to:

- Initiate and support legislation in the 98th Congress to amend appropriate sections of the Social Security Act to conform to the AMA definition of physician.

AMA Public Education Program

The House called upon the AMA to develop a model public education program for use by constituent societies. The program would deal with the potential threat to the quality of medical care from legislative proposals, *i.e.*, TEFRA and prospective payment systems.

Key Physician

The House urged state and county medical societies to develop with AMA assistance "key physician" contacts to aid the AMA staff in its Washington program. The House also commended the AMA Officers and staff, particularly the Washington staff, for their "superlative efforts on behalf of the FTC issue."

Anonymity/Resident Inquiries

The House adopted a report that guarantees anonymity of a resident physician who initiates an inquiry by a residency review committee into the conduct of a residency program. The procedure incorporates two basic principles:

- That residents should have available a process that allows them to bring forth concerns without fear of reprisal.
- That residency program directors and their teaching staffs should be protected from spurious complaints.

Residency Positions

The House adopted a report from the Council on Medical Education concluding that a sufficient number of residency positions are available in the Matching Program to accommodate current graduates of U. S. medical schools.

- However, there is *no assurance* that all U. S. graduates will obtain their *choice of specialty and/or location*.
- The report says that first-year enrollment in U. S. medical schools declined for the first time in 20 years of rapid expansion.
- This decline may be a response to the general perception that continued increase in output is no longer a national need.
- Financial limitations may be responsible for this trend.

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Legal Counsel

The House voted to encourage hospital medical staffs to consult with their own attorneys or those of their county medical society, state medical society, and/or AMA in securing knowledgeable legal counsel as appropriate.

Confidentiality in Review

The House voted to encourage medical staff peer review committees when evaluating the professional practices of fully licensed physicians to consider excusing non-physician members of the committee.

Prescription Drugs Advertising

The House adopted a report recommending that:

- The Food and Drug Administration act promptly to review the issue of advertising prescription drugs to the public.
- This advertising be held in abeyance pending the FDA's review.

The report cautioned that

Prescription drug advertising, whether to the health professions or to the public, that is false or misleading or does not give adequate information on beneficial as well as adverse effects is potentially dangerous and should be opposed.

Therapeutic Substitution

After much discussion including testimony from pharmacists' organizations in the reference committee, the House adopted a straightforward policy on therapeutic substitution. It reads: "That the AMA vigorously oppose any concept of therapeutic substitution of drugs by pharmacists."

Education Re: Dioxin

The "whereas" portion of this resolution was given widespread media attention; however, the House Action called on the AMA to:

- Institute an active public information campaign to get accurate information on dioxin before the public.

- Update a 1981 report on the subject.

Autopsies

The House adopted a resolution calling on the AMA:

- To develop and distribute materials to students and residents stressing the importance and necessity of the autopsy.
- To provide guidelines for obtaining medical/legal consent from families for the performance of this procedure.
- To investigate means of encouraging a higher proportion of autopsies.

AMA Dues

The House voted to raise the regular dues by $15, and $5 for medical students. Resident physician dues were not increased. The Board reduced its anticipated dues increase because of the current financial strength of the Association and the Board's desire to increase membership in all categories. In 1984 dues levels will be:

- $330 — Regular members
- $248 — Physicians in their second year of practice
- $220 — Physicians in military service
- $165 — Physicians in their first year of practice
- $45 — Physicians in residency training
- $20 — Medical students

Freestanding Emergency Medical Care Centers

The House adopted as amended a report prepared by the Commission on Emergency Medical Services that presented initial criteria to aid in determining whether a practice can truly offer a full range of emergency medical services. The criteria included:

- Hours of operation
- Staffing and medical direction
- Relationship to the emergency medical services system
- Ancillary services and equipment
- Protocols
- Private physician referrals
- Medical records
- Payment for services

Federal Funding for Ambulatory Care Centers

The House adopted a report that recommended more aggressive implementation by HHS and HCFA of existing provisions in federal legislation calling for equity of reimbursement between services provided by hospitals on an outpatient basis and similar services in physicians' offices.

Boxing as a Health Hazard

The House adopted a resolution that calls on the AMA to:

- Publicize the deleterious effects of boxing on the health of participants.
- Encourage the elimination of boxing from amateur scholastic, intercollegiate, and governmental athletic programs as detrimental to the health of participants.
- Develop model legislation seeking to curtail the utilization of boxing as a public spectacle to the extent feasible.

Conclusion

AMA House meetings provide a unique educational opportunity and we would encourage you to attend and participate. Any member of the Association may present testimony at the Reference Committee hearings. If you are unable to attend the meeting, you can still be represented through your delegates. Let us know your opinions. You can also prepare a resolution and request that it be submitted to the House. Many AMA policies began with an individual physician who had a good idea and coaxed it through the democratic process.

Thank you for giving us this opportunity to present this report.

Lew W. Purinton, M.D., *Delegate*
Alex Scott, M.D., *Delegate*
Kermit G. Wedel, M.D., *Delegate*
Jimmie A. Gleason, M.D., *Alternate*
Warren E. Meyer, M.D., *Alternate*
Linda D. Warren, M.D., *Alternate*

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Discontinuation of Valium (or Valrelease) is typically as smooth as its start in short-term therapy. However, Valium and Valrelease should be discontinued gradually after more extended treatment. As you diminish dosage, the built-in tapering action of Valium and Valrelease will help avoid rapidly recurring anxiety symptoms and symptoms of withdrawal, and will help ease the patient's transition to independent coping when therapeutic goals have been achieved.

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Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety. Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic. Symptomatic relief of acute agitation, tremor, impending or acute delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in: relief of skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome. *Oral forms* may be used adjunctively in convulsive disorders, but not as sole therapy. *Injectable form* may also be used adjunctively in: status epilepticus; severe recurrent seizures; tetanus; anxiety, tension or acute stress reactions prior to endoscopic/surgical procedures; cardioversion.

The effectiveness of diazepam in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindications: Tablets or capsules in children under 6 months of age; known hypersensitivity; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence.

> ***Usage in Pregnancy:*** **Use of minor tranquilizers during first trimester should almost always be avoided because their use is rarely a matter of urgency and because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

ORAL: Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral forms adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures.

INJECTABLE: *To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling and, rarely, vascular impairment when used I.V.: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veins,* i.e., *dorsum of hand or wrist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Injectable Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.*

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea; have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs.

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status. Not recommended for OB use.

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of diazepam, *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed and tolerated).

The clearance of diazepam and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

INJECTABLE: Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated.

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug.

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, observed in patients during and after diazepam therapy are of no known significance.

INJECTABLE: Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia.

In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported.

Dosage: Individualize for maximum beneficial effect.

ORAL: *Adults:* Anxiety disorders, relief of symptoms of anxiety—Valium (diazepam/Roche) tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 Valrelease capsules (15 to 30 mg) daily. Acute alcohol withdrawal—tablets, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; or 2 capsules (30 mg) the first 24 hours, then 1 capsule (15 mg) daily as needed. Adjunctively in skeletal muscle spasm—tablets, 2 to 10 mg t.i.d. or q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily. Adjunctively in convulsive disorders—tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily.

Geriatric or debilitated patients: Tablets—2 to 2½ mg 1 or 2 times daily initially, increasing as needed and tolerated (see Precautions). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose.

Children: Tablets—1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use in children under 6 months). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose (not for use in children under 6 months).

INJECTABLE: Usual initial dose in older children and adults is 2 to 20 mg I.M. or I.V., depending on indication and severity. Larger doses may be required in some conditions (tetanus). In acute conditions injection may be repeated within 1 hour, although interval of 3 to 4 hours is usually satisfactory. Lower doses (usually 2 to 5 mg) with slow dosage increase for elderly or debilitated patients and when sedative drugs are added. (See Warnings and Adverse Reactions.)

For dosages in infants and children see below; have resuscitative facilities available.

I.M. use: by deep injection into the muscle.

I.V. use: inject slowly, take at least one minute for each 5 mg (1 ml) given. Do not use small veins, i.e., *dorsum of hand or wrist. Use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.*

Moderate anxiety disorders and symptoms of anxiety, 2 to 5 mg I.M. or I.V., and severe anxiety disorders and symptoms of anxiety, 5 to 10 mg I.M. or I.V., repeat in 3 to 4 hours if necessary; acute alcohol withdrawal, 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary. Muscle spasm, in adults, 5 to 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary (tetanus may require larger doses); in children *administer I.V. slowly*; for tetanus in infants over 30 days of age, 1 to 2 mg I.M. or I.V., repeat every 3 to 4 hours if necessary; in children 5 years or older, 5 to 10 mg repeated every 3 to 4 hours as needed. Respiratory assistance should be available.

Status epilepticus, severe recurrent convulsive seizures (I.V. route preferred), 5 to 10 mg adult dose administered slowly, repeat at 10- to 15-minute intervals up to 30 mg maximum. Repeat in 2 to 4 hours if necessary, keeping in mind possibility of residual active metabolites. Use caution in presence of chronic lung disease or unstable cardiovascular status. Infants (over 30 days) and children (under 5 years), 0.2 to 0.5 mg slowly every 2 to 5 min., up to 5 mg (I.V. preferred). Children 5 years plus, 1 mg every 2 to 5 min., up to 10 mg (slow I.V. preferred); repeat in 2 to 4 hours if needed. EEG monitoring may be helpful.

In endoscopic procedures, titrate I.V. dosage to desired sedative response, generally 10 mg or less but up to 20 mg (if narcotics are omitted) immediately prior to procedure; if I.V. cannot be used, 5 to 10 mg I.M. approximately 30 minutes prior to procedure. As preoperative medication, 10 mg I.M.; in cardioversion, 5 to 15 mg I.V. within 5 to 10 minutes prior to procedure. Once acute symptomatology has been properly controlled with injectable form, patient may be placed on oral form if further treatment is required.

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension. Dialysis is of limited value.

How Supplied:

ORAL: Valium scored tablets—2 mg, white; 5 mg, yellow; 10 mg, blue—bottles of 100 and 500; Prescription Paks of 50, available in trays of 10; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25 and in boxes containing 10 strips of 10.

Valrelease (diazepam/Roche) slow-release capsules—15 mg (yellow and blue), bottles of 100; Prescription Paks of 30.

INJECTABLE: Ampuls, 2 ml, boxes of 10; Vials, 10 ml, boxes of 1; Tel-E-Ject® (disposable syringes), 2 ml, boxes of 10. Each ml contains 5 mg diazepam, compounded with 40% propylene glycol, 10% ethyl alcohol, 5% sodium benzoate and benzoic acid as buffers, and 1.5% benzyl alcohol as preservative.

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Tenth Annual UKSM — Wichita Issue

The Dean's Letter — 1983

The University of Kansas School of Medicine-Wichita

WILLIAM J. REALS, M.D., *Wichita*

We are pleased once again to present this annual scientific portion of the September 1983 issue of *The Journal of the Kansas Medical Society*. Several of our faculty members have prepared scientific papers of broad interest and of high scientific quality reflecting the work of our faculty in medical education in the Wichita area.

We are now in the second phase of the project to convert our building into the UKSM-Wichita academic center. This is a remodeling of the interior for new offices, classrooms, and other educational activities. A new completely remodeled outpatient clinic also is in this phase and will be in use later this year. The entire project cost is $4.3 million, and we hope that by the end of 1984 the building program will be completed.

Each July, upon completion of their second year at the KU Medical Center, 50 medical students begin clinical training at Wichita and on our campus. Many of their classes and other activities are held in our four affiliated hospitals: The Veterans Administration Medical Center, St. Francis Regional Medical Center, Wesley Medical Center, and St. Joseph Medical Center. These hospitals and their staff fully support our medical school, and without their cooperation the school could not exist. We are very grateful for all services provided to us by the Wichita hospitals both in medical education and graduate medical education. Our graduate programs are hospital-based and encompass programs in most of the major medical specialties including three programs in the city in Family Practice. A fourth program in Family Practice is located at Salina on the campus of Kansas Wesleyan University; it is sponsored by the Salina Health Education Foundation and the University of Kansas School of Medicine.

More than 430 volunteers hold teaching appointments on our campus in addition to 37 full-time, and 104 part-time faculty. The Medical School staff of 124 support our educational programs. These dedicated individuals maintain our building, assist in treatment of patients in our outpatient clinic, monitor progress of our students, and provide the many other necessary services to our medical education programs.

UKSM-W is a community-based school utilizing the resources of Wichita and the surrounding area. It is one of the most successful examples of the new concept of a university without walls. The school could not function without the wholehearted support of the medical and hospital community in Wichita, and we express our gratitude to them for their continuing involvement and interest in both undergraduate and graduate medical education. We have also enjoyed outstanding support from our colleagues at The University of Kansas Medical Center in Kansas City and The University of Kansas in Lawrence.

Our school owes a great debt of gratitude to Dr. David Waxman, the retiring Executive Vice Chancellor of UKSM, who has been involved in the development of UKSM-W almost from its beginning. His leadership and encouragement have materially assisted the faculty as well as the staff and others associated with our school. We wish him well in his new position as Director of Health Care Resources for UKSM.

Our student body, faculty, and staff all join me in expressing gratitude to all who have made our school possible. We pledge our continuing support in providing health care to the citizens of Kansas.

The View From the Bridge

A Decade of History at the University of Kansas Medical Center

DAVID WAXMAN, M.D., ***Kansas City, Kansas***

THE ANALOGY of a bridge in the title of this talk is fitting, for it connotes continuity, obstacles to be overcome, transition, and new paths to follow — all relevant to experiences at the University of Kansas Medical Center in recent years. The analogy is appropriate in still another sense, for the bridge of a ship affords a vantage point for viewing operations and the environment in which they occur. It is where plans are made — plans that are shaped by information from several sources; plans that affect the security and performance of the crew; plans that ultimately have an impact upon the progress of the ship in pursuit of its mission. Like the conceptual bridge, the ship's bridge offers a special perspective — a view of how disparate elements relate to each other and of how an immediate situation relates to a broader scenario. This message is presented from that perspective of the University of Kansas Medical Center. It will focus on events of the past ten years within the context of earlier developments.

When I joined the faculty in 1961, the institution was run by a dean, an assistant dean, the department chairmen, a hospital administrator, a registrar, and a fiscal officer. There was only one professional school — the School of Medicine — which included a Department of Nursing and several allied health technician training programs. Today we are a College of Health Sciences and Hospital with Schools of Medicine, Nursing, Allied Health, and Graduate Studies. We now have an executive vice chancellor, two vice chancellors, six deans, several associate and assistant deans, a hospital administrator, and a plethora of new courses of study and programs that take place on two campuses.

Life was comparatively simple then. A full, four-year accreditation had been awarded to the School of Medicine, and all basic science programs were being consolidated at the Kansas City campus. A long range plan for physical growth of the Medical Center had been submitted to the Board of Regents in 1960. It included an expanded outpatient facility, new offices and patient rooms, pediatric wings, and dormitory rooms in the Student Center. Most of these projects were completed by 1965.

In 1961, there were 762 students here, 292 of whom were enrolled in the School of Medicine. Approximately 148 interns and residents were enrolled in housestaff programs, and about 322 students in nursing, allied health, and graduate programs. During the next 22 years, the number of students increased by nearly 350 per cent — to more than 2500 students in 1983. All schools in the College of Health Sciences contributed to that remarkable growth.

The rise in student enrollment is only one indicator of change brought about by public policy decisions in the late 1950s. Let us briefly review some of the societal forces that were to have such a profound impact, for they are relevant to recent history here and to our future directions.

The first federal legislation in direct support of health care education was enacted in 1963 — the Health Professions Educational Assistance Act. It triggered a broad array of initiatives to address the problems of shortages and maldistribution of health care providers. Federal programs were accompanied by a variety of state, local, and private initiatives.

These initiatives evoked a number of changes.

- The number of accredited medical schools expanded from 79 in 1950 to the present 126.
- U. S. medical school enrollments increased nearly 150 per cent, from about 26,000 in 1950 to more than 65,000 in 1982.
- Affirmative action admission guidelines changed the composition of the student body to include more women and minorities. Women now make up nearly 30 per cent of first year classes.
- The full-time medical faculty grew from 11,200 in 1960 to nearly 50,000 in 1981.
- Costs of health sciences education soared. In 1960, U. S. medical schools received about $436 million in financial support from all sources. In 1980, support had grown to more than $4.9 billion.

This material has been adapted from the Lecture of the Sterling Drug Visiting Professorship, June 29, 1983. The lecture is sponsored by Sterling Drug Company, whose Chairman of the Board, W. Clarke Wescoe, formerly served as Dean of the School of Medicine and Chancellor of the University of Kansas.

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- Federal involvement extended into nearly every aspect of health professions education — class size, curricula, residency programs, student loans, research, and construction.
- The last 20 years have seen: the rise and fall of the three-year medical program; additional required and elective courses; advances in instructional design, technology, and classroom management; expanded roles for students in decision-making; and greater attention to the evaluation of faculty performance.
- Primary care medical specialties have reached new heights of importance as evidenced by the growth in residency positions in pediatrics, general internal medicine, and family practice. It is now estimated that more than 60 per cent of each graduating class enters residency programs in these three specialties alone.
- There has been significant expansion of education and career opportunities in all health professions, especially in nursing, dentistry, and the allied health areas. New schools were developed and new degree programs proliferated to parallel changes taking place in medical education.
- Undergraduate, graduate, and continuing education have been increasingly conducted at locations distant from the health sciences campus. These decentralized educational activities are best illustrated by the Area Health Education Center (AHEC) programs.

The latency and impact of the problems described earlier and the new initiatives to resolve them varied from one place to another. At the University of Kansas, they reached a critical mass by the early 1970s. At about the same time, another societal development — increasing competition and commercialism in health care services — was making its mark on university medical centers. The university hospital could no longer rely on prestige, tradition, and referrals from community physicians to keep its beds occupied, revenues up, faculty content, and educational programs intact. University hospitals and their associated medical schools had, in one sense, done their job too well. Their graduates — the physicians who received specialty training in their classrooms and patient areas — were now caring for the very patients who, at another time, might have been seen at the university hospital. Private hospitals developed their own capability to provide specialty care. New hospitals were built, bed numbers were increased, the most recent technologies were acquired, and tertiary care centers multiplied. Patients and referring physicians utilized the nearest facility that was capable of providing the required care. It was during this period of burgeoning health care resources that health planning agencies appeared.

To some segments of the health care community, these trends had been apparent for several years, but for the most part, university medical centers either ignored them or downplayed their significance. By the early 1970s, however, the declining patient census at university hospitals could no longer be ignored. Problems intensified during subsequent years to the extent that the university hospital has become an endangered species.

At a highly visible, public institution, the University of Kansas Medical Center means different things to different sectors of society. Events occurring at the Medical Center during the past decade have been interpreted from their own perspective by legislators; public officials; the federal government; practicing physicians and other health professionals; lawyers; the news media; patients; ambulance drivers; insurance companies; other educational institutions; and our own students, employees, faculty, and alumni. It is intimidating and humbling to consider the vast number of lives we touch.

During the past decade as dean, vice chancellor and executive vice chancellor, it was my privilege to relate to nearly all groups mentioned above and to many others as well. I have been an advocate, interpreter, and defender for the University of Kansas Medical Center. I have had an opportunity to serve the University and the State of Kansas; I confess to a deep and abiding love for each. We have been through difficult times and have had serious problems, but we must keep in mind the larger context within which they occurred. The entire field of health care has been prominent in the public eye for some time. Health care professionals and educators have not only been agents *for* change; they have been targets *of* change. Is it any wonder that health professional schools would resonate to those social and economic currents?

To those who feel that we have been in a stormy or controversial period, I can only paraphrase the words of Longfellow: "Troubled times are but a mist-covered bridge leading from Light to Light, through a brief darkness." From another perspective, as we look back upon the last decade or so, we have achieved much of which to be proud.

Our new physical facilities include: Bell Memorial Hospital; Orr-Major Hall; the Mid-America Radiation Therapy Center; Applegate Energy Center; and the multilevel parking facility. There has been an extensive renovation of office and patient care areas in older portions of the hospital, and Battenfeld Auditorium has been improved. Con-

tinuing efforts in housekeeping and landscaping have greatly enhanced the appearance of the complex.

We will soon move our library collection into the new Archie R. Dykes Library of the Health Sciences, and this attractive and functional building will give a tremendous boost to the learning environment at the Medical Center.

It is easy now to forget the nuisances that annoyed us for nearly six years. From that frustration, the face of this campus has been transformed, a physical plant has been created that better equips us to carry out our mission of education, research, and patient care. The faculty and staff have made a superb contribution to many activities, especially in effecting the move into the new hospital. It was a monumental coordinated effort involving patients, equipment, supplies, and personnel that was carried out almost flawlessly.

Important, if less obvious, are recent operational and programmatic achievements. In response to pressures for additional health professionals for Kansas, we increased our student population and our faculty size. Schools of Nursing and Allied Health were created; a Department of Family Practice was established; and significant additions were made to all residency programs. The Division of Health Care Outreach was formed to assist small communities in their efforts to meet local health care needs. The area health education centers in Hays, Garden City, and Chanute have gained widespread recognition, and with the Outreach offices in Kansas City and Wichita, they form a statewide network of educational service centers that link the resources of the Medical Center to communities throughout the state.

The increase in students and residents, the impact of the Kansas Medical Scholarship Plan, and the contributions of the outreach program have halted and reversed the decline in medical manpower in Kansas. Underserved areas continue to exist, but the situation has improved sufficiently so that the once unthinkable proposition to reduce the size of our entering medical school class is now under consideration.

In response to recent competitive pressures, we have pursued two goals of greater efficiency and an increased base of patient referrals. We have installed a computerized billing and accounting system; outpatient services are under a new operations format; improved support services now make it easier for patients to be evaluated and to find access to hospital resources; greater attention is paid to expectations of referring physicians; two satellite clinics have been developed in the outlying community; we have affiliated with a health maintenance organization — Kansas City Health Care, Inc.; and educational and service agreements have been established with military, Veterans Administration, and several state hospitals.

The Department of Radiation Therapy provides excellent services for cancer patients. The Departments of Ophthalmology and Otorhinolaryngology have moved into expanded and attractive new quarters. Promising new faculty have been recruited, and these departments are becoming regional referral centers.

The Department of Rehabilitation Medicine is scheduled to move into expanded and renovated quarters. Through the generosity of the Sutherland family and the Jones Trust, the Sutherland Microsurgical Center utilizes the latest techniques in microscopic surgery to achieve the replantation of limbs and digits, and to surgically correct pediatric deformities.

We are proud of the many improved and expanded hospital services. Hospital operations are still in a fluid state, subject to changes in reimbursement policies and procedures. We also continue to feel the pressure of competition from other hospitals and the threat of alternative management configurations. However, we are in a stronger position than ever before to respond adaptively to those pressures.

The new Senior Citizen Center is a good example of our expanded patient care. Bell Memorial Hospital is a recognized and vital component of our academic health science center and continues to progress under dynamic leadership.

Although our institutional energies have been influenced by external developments in recent years, not all of those energies should be attributed to the stimulus of external events. There is a natural evolution and growth in any institution such as ours, and they reflect the insight, intelligence, commitment, and initiative of its faculty, students, and staff. External events may have prompted some recent efforts, but they served more as catalysts for changes that would have taken place otherwise.

Our academic programs have made tremendous strides, with growth in faculty and programs at the College of Health Sciences and in the number and quality of our students. A five-year plan has been developed and is now being updated for the Board of Regents. Our Schools of Medicine and Nursing have been successfully accredited as have several programs in Allied Health. The School of Nursing has organized a program in graduate nursing outreach that is widely recognized as a national model, and its new Ph.D. program is being positively considered

for federal support. The School of Allied Health has consolidated its departmental structure and has grown impressively in its educational maturity, quality, and productivity. Still, Nursing and Allied Health have obstacles to overcome. They continue to seek improved instructional facilities and salary support, and these will doubtless be high priorities in the years ahead.

The KU Medical Alumni Association has generously supported medical education and the biomedical sciences through its Distinguished Medical Teaching Fund. This fund encourages activities that bridge the gap between the basic and clinical sciences. In recent years, support has been provided for visiting speakers as well as for the Departments of Anatomy, Biochemistry, and Physiology. Funding has recently been obtained to support the position of Chairman of the Microbiology Department.

It is impossible to overstate the importance of an active and productive research effort at an institution such as ours. Basic biological, behavioral, and clinical research contribute significantly to medical education. Progress in biomedical research is synonymous with better education and improved patient care. The environment for research here has improved considerably, and with sustained effort by the faculty and with support from the Chancellor, the Board of Regents, the Governor and the Legislature, it will doubtless continue to improve. It is essential that efforts be continued to better educate those we serve about the importance of the research that is conducted here.

In retrospect, the last decade has been very productive, even if at times we seemed to operate in the eye of a hurricane. During this time, the University of Kansas School of Medicine-Wichita was established as a community-based educational facility and has become an influential and significant component of that city. The campus, established in 1971, is now located at 1001 N. Minneapolis, and houses administrative offices, classrooms and outpatient clinics. In addition, the resources of three community hospitals and the Veterans Administration Medical Center contribute to the teaching of medical students and residents. Twenty-five per cent of the students from each entering medical school class in Kansas City complete their last two years of clinical training in Wichita. UKSM-W is currently involved in an extensive renovation program at its main facility. Planning for the final phase is well underway.

The development of a governance document for the Medical Center represents the outcome of almost three years of Wichita and Kansas City faculty energies. It was forged in an attempt to define the special considerations that apply to an academic health science center and is a significant achievement.

The governance document reflects still another area of progress in terms of improved communications among the various elements that make up the Medical Center community — faculty, students, administration, and support staff. Together we have broken down barriers of communication and established a healthier, freer, more constructive dialogue. The Interinstitutional Committee made up of departmental chairmen from Wichita and Kansas City and headed by our two medical school deans serves to bind our two campuses together academically, emotionally, and politically.

The state and county medical societies have provided us tremendous guidance and support through the years. A very important contribution by the membership and spouses is through the AMAERF activity. These monies directly aid our medical students and are vital during these times of loss of federal support. It is essential that all of us participate actively in medical society functions.

Any catalog of improvements here would be incomplete if it failed to recognize the contributions of the KUMC Auxiliary. This group has done much to improve conditions for the faculty, students, staff, patients, and visitors. Their latest undertaking — the JayCare Learning Center — provides a high quality of day care for children of our employees, thus improving our ability to attract and retain competent, qualified staff. We are grateful to the Auxiliary for developing and operating the Center, and to the KU Endowment Association for assistance in its initiation.

It has been my good fortune to serve as executive vice chancellor while many of these accomplishments — your accomplishments — were taking place. From the perspective of that position, I have witnessed a close scrutiny, sometimes stern criticism, but always firm support for this institution — support from the Board of Regents, from the governor, and from the Kansas legislature. We are in a much better fiscal position than many state-supported health science centers, and the main reason is the people of Kansas; through their elected and appointed representatives, state appropriations to the Medical Center have increased dramatically during the past decade.

We have been fortunate in attracting tremendous support from our state, support that reflects confidence in the University of Kansas. We have passed through an era of building to enter an era of consolidation and reinforcement. There is much room

(Continued on page 449)

Departmental Report

Division of Community Medicine, Department of Family & Community Medicine

ANNE D. WALLING, M.D., Chairman

The Department of Family and Community Medicine at The University of Kansas School of Medicine-Wichita has continued its growth and development during the past four years. Its administrative structure and faculty requirements are now well established to meet the needs of family medicine at UKSM-W, despite considerable fiscal constraints imposed by the depressed economy. The department has accepted responsibility in the academic areas of scholarly activities, research, and service.

Currently, department faculty consists of 17 full-time, 24 part-time, and 60 volunteer physicians from Sedgwick and neighboring counties. The administrative staff now includes: Chairman; Administrative Assistant; Vice Chairman of Undergraduate Education in Family Practice; Head of the Division of Community Medicine; Head of Behavioral Sciences in Health Care; Head of Research and Development; with an unfilled position of Head of Postgraduate Studies. Faculty with direct responsibility for our graduate (residency) programs consist of: Wesley Medical Center — a Program Director and three Associates; St. Joseph Family Practice Residency Program — a Senior Associate Program Director and three Associates; Salina Family Practice — a Program Director, one Associate, and one Assistant; and St. Francis Regional Medical Center — an Acting Program Director, one faculty member, and two unfilled faculty positions.

The curriculum for undergraduate students consists of a one month clerkship in the junior year and a one month rural experience in the senior year. Electives will be offered in community medicine and family practice. Attempts will be made to increase the curricular time for implementation of geriatric and community medicine as a component part of our curriculum. Students in their third year gain experience with family physicians in the Wichita urban area and also in our family practice residency centers at Wesley, St. Joseph, Salina, and St. Francis. A one month rural experience is now offered in the four rural teaching sites of Harper, Greensburg, McPherson, and Sterling.

We now have 74 residents in training in the four divisional family practice residencies of Wesley, St. Joseph, St. Francis, and Salina. We have had excellent results in recruiting Kansas students to our residencies and in retaining our residents in Kansas and the immediate perimeter area; during the last four years, 67 per cent remained in Kansas to set up practice.

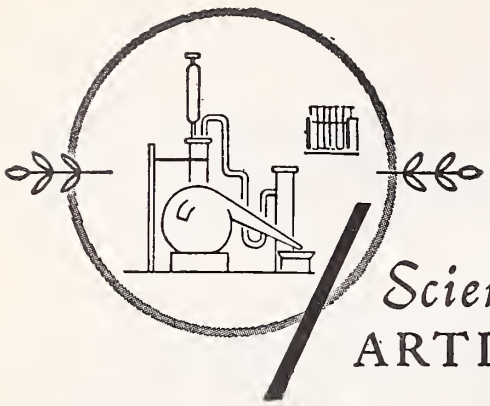
Continuing Medical Education for the practicing physician has received considerable emphasis. We have worked closely with the Kansas Academy of Family Physicians to identify specific needs and to develop programs to meet those needs. Future activities will include: The Second Annual Family Practice Program, "The Skin: Covering the Subject"; "The Family and Medicine"; and a Cardiology Traineeship in conjunction with the American Heart Association.

Another program will teach improved educational skills to our faculty. Our Annual Shaw Memorial Lectureship will be presented for the first time this year, and the Ernie Chaney Postgraduate Miniseminar series will also be implemented.

Research efforts have been enhanced by the addition of a Director of Research and Development and a Head of Community Medicine. The installation of a new computer at the Medical School has enabled us to begin storing data for applied investigative research in Family Practice. A series of seminars on the development of skills in investigative studies has recently been initiated for physicians in Kansas and the immediate perimeter area.

Investigative studies are being conducted in the area of perinatology and infant and fetal mortality by members of the Department in conjunction with the Wichita/Sedgwick County Health Department.

The Department has made significant progress in achieving the goals and objectives developed for meeting its responsibilities at UKSM-W. The future of the Department and activities will be directly related to state needs for family physicians, to the state economy, and to the University of Kansas administration.



Riley-Day Syndrome

Management of Anesthesia for Patients With Familial Dysautonomia

TIMOTHY T. FREEMAN, M.D.*; SHELBY JESTER, M.D.† *and* KARL E. BECKER, JR.‡, M.D., *Wichita*

Familial dysautonomia (Riley-Day syndrome) is an uncommon systemic disorder of the autonomic nervous system typically affecting Jews of Ashkenazic origin. Transmission is through an autosomal recessive genetic pattern giving an incidence among North American Jews of 1 in 10,000 to 1 in 20,000 live births.[1] The disease is manifest by a pronounced insensitivity to pain and temperature, alacrimation, dysphagia with frequent aspiration, postural hypotension, and labile hypertension with episodic fever and vomiting. These patients also suffer from emotional lability and absent taste sensation.

In 1959, Kritchman *et al.* first described the problems of administering general anesthesia to patients with familial dysautonomia.[2] In that series of 27 anesthetics given to eight patients, severe hypotension occurred six times and cardiac arrest twice. The authors concluded that thiopental and tribromoethanol were responsible for these precipitous reactions. Likewise, McCaughey reported that ether, halothane, and methoxyflurane all produced hypotension in a child with dysautonomia.[3]

The case presented here illustrates a satisfactory method of anesthetic management for a minor orthopedic procedure on a child with familial dysautonomia.

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Case Report

An 8-year-old, 21 kg male of distant Jewish descent presented with analgesic body and limbs, decreased temperature sensation, scoliosis, incontinence, frequent pulmonary and skin infections, and corneal scarring. Family history was negative for dysautonomia. Past history described feeding problems since infancy, episodic fever, incoordination, bowel disturbances, episodic vomiting, and emotional lability. Physical examination revealed an alert, cooperative, however somewhat anxious child. Vital signs were blood pressure 100/60; pulse, 80; temperature, 36.8C.; and respirations, 24. The child was drooling and showed evidence of a minor speech defect. Pupils were small and corneal scarring was present on the left. The tongue was smooth. Results of auscultation of the heart and lungs were unremarkable. Numerous burns and abrasions were present over the trunk and extremities which showed marked insensitivity to pain and temperature. The lower back was scoliotic. The hematocrit was 37.8% with 9200 white blood cells and an estimation of

Familial dysautonomia (Riley-Day syndrome) is a systemic disorder of the autonomic nervous system. This discussion focuses on the physiologic basis of the disease and the management of anesthesia for patients with such disorders.

normal platelets. A bone scan revealed evidence of osteomyelitis of the left middle finger. The patient was scheduled for incision and drainage of that finger.

The child was brought to the operating room without premedication where blood pressure cuff, EKG, and precordial stethoscope were applied. Chlorpromazine, 5 mg, was given intravenously and repeated 20 minutes later. Gentle inhalation induction per mask with 65 per cent nitrous oxide in oxygen was performed without incident. A temperature probe was placed in the right axilla where it recorded 35C throughout the operation. Respirations were spontaneous and blood pressures remained at 130/90 throughout the case, rising briefly at emergence to 150/105. Heart rate was a constant 76/minute, except during induction when it rapidly increased to 120 for one minute, then quickly slowed again to 76. At the conclusion of the operation, 100 per cent oxygen was given, and the child awakened without incident. Total anesthesia time was 65 minutes.

The child was returned to the operating room three days later for secondary closure of his left hand. Similar techniques were used for this 45-minute procedure, including chlorpromazine, 7.5 mg intravenously in divided doses, and 65 per cent nitrous oxide. The anesthetic course was again uneventful and awakening at the conclusion of the operation was rapid.

Discussion

The physiological basis of familial dysautonomia is not fully described; however it appears that there is an imbalance of the autonomic, sensory, and central nervous system. This is manifest as an abnormally low parasympathetic system activity with episodes of intense outbursts from the sympathetic system. Consistent anatomic findings are lacking but the peripheral nerve has been implicated on the basis of abnormal reactions to intradermal histamine. Invariably in dysautonomic patients, a wheal forms but there is no surrounding flare. This has been used as a screening test for familial dysautonomia. Other peripheral nerve studies have shown decreased unmelinated nerve fibers[4] and fewer neurons in autonomic ganglia.[5]

Smith and Dancis[6] have reported three other features of dysautonomia including abnormalities of catecholamine metabolism, abnormal sensitivity to norepinephrine, and sensitivity of the pupil to conjunctival injection of dilute acetyl-beta-methylcholine. During sympathetic nerve stimulation, dopamine-beta-hydroxylase is released with norepinephrine in a constant ratio such that plasma dopamine-beta-hydroxylase levels can be a marker of catecholamine release during standing or exercising. In dysautonomic patients these levels are markedly decreased in comparison to normal subjects with intact autonomic nervous systems.[7] This chronic insufficiency of catecholamines at the receptor sites may explain the supersensitivity of these patients to infused norepinephrine.

In planning the anesthetic management of patients with familial dysautonomia, several features should be considered. Premedication is advised since often these children come to the operating room in an apprehensive state. Chlorpromazine and meperidine have both been used successfully.[8] Normal cardiovascular responses to changes in blood volume may not occur; therefore careful preoperative assessment of fluid status and accurate intraoperative estimation of blood loss is required. Thiopental inductions should probably be avoided, since severe bradycardia and hypotension have been reported.[2] Potent inhalation agents have also led to significant hypotension, although Meridy and Creighton[9] successfully used halothane in concentrations of 0.5 per cent or less. Temperature monitoring is necessary to detect sudden hyperthermia that may occur in the absence of sweating and labile sympathetic tone.

Minor peripheral procedures that are, in normal patients, quite painful may be well tolerated with little or no anesthesia in dysautonomic patients due to markedly diminished pain sensitivity.[10] Thus, as in our case, the goal of anesthetic management is to provide a degree of sedation that allows the operation to be performed. Chlorpromazine provides that sedation and also may help prevent the nausea and vomiting that these patients frequently develop. The alpha-adrenergic blocking effect of chlorpromazine may be a disadvantage; however, Meridy and Creighton[9] have described its use without hypotensive episodes and hypotension was not a problem in this case.

The absence of normal gag reflexes in patients with familial dysautonomia is a feature that leads to much of the morbidity and mortality associated with this disease. In this case our anesthetic technique provided sedation that allowed the patient to maintain adequate spontaneous respirations and control of his airway. The technique described in this case appears to be a safe and simple method of anesthetic management for the patient with familial dysautonomia.

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Positive Pressure Ventilation

Respiratory Effects on EKG Axes

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IT IS COMMON medical knowledge that the EKG axes can vary with respiration. Deep inspiration causes the heart to assume a more vertical position and greater clockwise rotation. Deep expiration causes the heart to become more horizontal.[1]

The two patients reported here experienced marked and transient EKG axis changes when undergoing major surgery.

Case Reports

EKG leads were placed on the back of each patient over each upper shoulder and each lower flank. Leads I, II, and III were monitored. After induction patients were placed on a ventilator with a volume of 10 ml/kg.

Case One

A 65-year-old female scheduled for carotid endarterectomy and coronary artery bypass grafts was induced with fentanyl, diazepam, and pancuronium. Prior to the endarterectomy, a sternotomy was performed but the pericardium was not opened. EKG at this time showed a marked QRS axis shift from −5 degrees during expiration to +65 degrees during inspiration. This shift was most marked in lead III where there was a complete reversal of polarity *(Figure 1)*. Standard 12 lead EKGs revealed a mean QRS axis of −5 degrees preoperatively and −15 degrees postoperatively.

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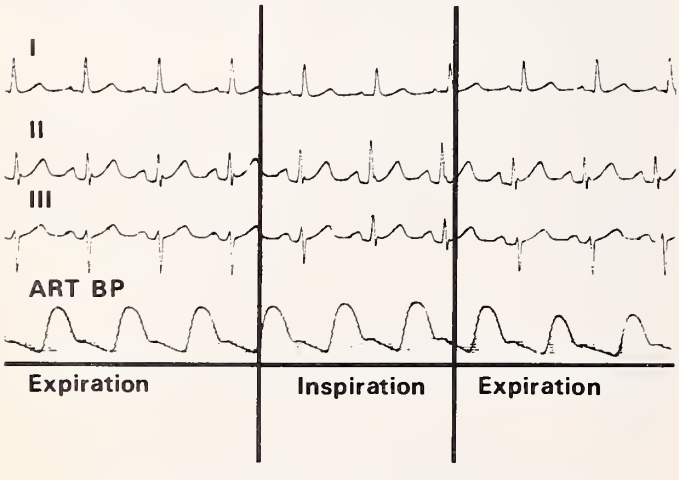


Figure 1. Electrocardiogram, Case One.

Two cases of marked shifts in mean QRS axes and complete reversals of polarity in lead III during positive pressure ventilation in paralyzed patients are presented. Causes of such shifts in EKGs are discussed.

Case Two

A 78-year-old female with chronic renal failure and CHF was maintained with nitrous oxide, fentanyl, and curare for an exploratory laparotomy. EKG during the case showed a QRS axis shift from +25 degrees during expiration to +65 degrees during inspiration. This shift was most noticeable in lead III where a complete reversal of polarity was noted *(Figure 2)*. Standard 12 lead EKGs revealed a mean QRS axis of +35 preoperatively and +47 degrees postoperatively.

Discussion

Deep inspiration and expiration can alter the axes of EKGs. With inspiration the heart becomes more vertical and there is greater vertical displacement; with expiration the heart becomes more horizontal and there is greater horizontal displacement.[1] However changes in cardiac position may be an oversimplification in explaining EKG axis shifts. In addition, deep inspiration and expiration can cause variations in stroke volume, ventricular volumes, and lung conductivity[2] as well as the center of gravity of the body.[3] Measurements of intracardiac volumes during inspiration show right ventricular enlargement due to negative intrathoracic pressures.[4] The left ventricle receives less blood, and the pulmonary vasculature expands. On expiration the opposite effects occur. During positive pressure ventilation the sequence reverses. As the left ventricular stroke volume decreases, the termi-

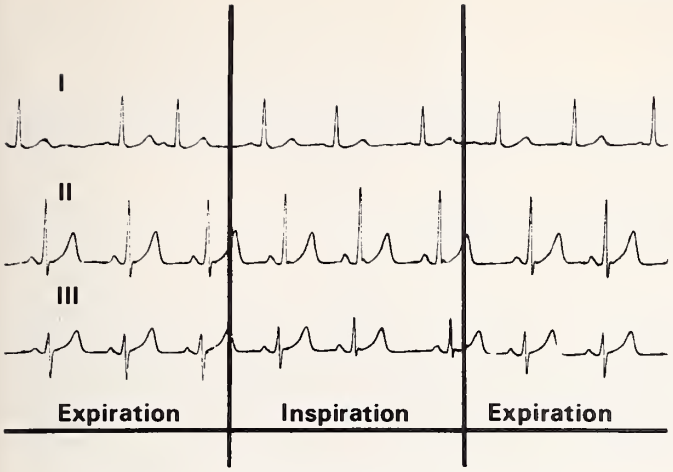


Figure 2. Electrocardiogram, Case Two.

nal QRS spatial vector decreases. Respiration has been noted to affect primarily the terminal half of the QRS cycle. Thus the changes noted in the EKG with respiration correlate with known changes in the volume of the ventricles.

In these cases, the change in mean QRS axis from expiration to inspiration was 70 degrees in the first case and 40 degrees in the second. This was especially noticeable in lead III where there was complete reversal of polarity of the QRS complex. This was due to positive pressure ventilation causing vertical displacement of the heart in these paralyzed patients and accompanying ventricular volume changes.

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The View from the Bridge

(Continued from page 444)

for continued, even dramatic growth, but growth conducted in an atmosphere that is characterized less by crisis than by careful, deliberate planning. We should:

- Strengthen our educational programs to better prepare our students for their chosen careers.
- Continue to improve the competitive posture of the hospital.
- Enhance our research potential and productivity.
- Devote greater attention to the expectations and needs of referring physicians.
- Increase stipends for housestaff.
- Assess the level of student enrollment in light of changing needs in health manpower and in the employment situation.
- Sustain a course of progress in clinical affairs so that our patient care programs lead those available elsewhere.
- Reallocate existing resources from areas of lesser need to those of greatest need.
- Adjust faculty salaries based on individual productivity.
- Obtain greater support for the acquisitions budget of the health sciences library.
- Construct a physical fitness/recreational center on the campus.
- Expand our current efforts so as to achieve the maximum possible conservation of energy.

These challenges are in no particular order. They should guarantee an exciting and promising future for the University of Kansas Medical Center.

We are fortunate to be the recipients of one of the finest blessings that society can offer — the opportunity to engage in socially meaningful labor in a rational, orderly, protected, and rewarding environment. We are in a position to attract praise, admiration, and respect. We have our detractors, but even when others seem disappointed *in* us, it is often a gauge of how much they expect *from* us.

We serve a greater community and a greater purpose than most are privileged to do. We must never take that for granted. In this age of ever increasing technology and ultra-specialization, we run the risk of losing those precious commodities we were taught early to cherish — common sense and a sense of humor. Our decisions as educators and as professionals must keep the human element clearly in focus. We must reflect on the meaning of what we do and how it may affect the lives of others.

The future will require some hard decisions. But those decisions must be made in consideration of the security and performance of the crew — faculty, students, and support personnel — and the progress of the ship — The University of Kansas Medical Center — as it pursues its mission. Decisions that affect the life of an institution must often transcend immediate events and personal concerns and must take into account the view from the bridge.

Fetal/Infant Mortality

A Review of Sedgwick County Statistics for 1981

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PUBLICATION of the infant mortality statistics for the State of Kansas for 1981 revealed an unexpected increase in the number of infant deaths reported for Sedgwick County. Considerable local publicity resulted, expressing community concern regarding Sedgwick County's high infant mortality rate.[1] This increase in the number of infant deaths is a particular cause for concern since the perinatal services available in Sedgwick County are among the most extensive and sophisticated in the state.

Although the Sedgwick County infant mortality rate has been consistently higher than the state average for several years, it had been falling slowly until 1981, when it rose from 11 to 13.2/1000. A comparison of the Kansas and Sedgwick County infant mortality figures for 1977-1982 is given in *Table I*. Although the 1981 figures do not represent a statistically significant increase in local infant mortality, an analysis was conducted by the Wichita/Sedgwick County Health Department and the Division of Community Medicine, Department of Family and Community Medicine, The University of Kansas School of Medicine-Wichita.

Definitions

Kansas statute defines *infant death* as the death of a live-born infant under one year of age with no minimum weight or gestation criteria. *Fetal death* (or stillbirth) is defined in Kansas as a product of conception weighing more than 350g that is not a live birth. Intentionally aborted fetuses are not included in the definition, nor are stillbirths weighing less than 350g. In 1963, the minimum weight criterion of 350g replaced the previous standard of 20 weeks gestation in the definition of fetal death.

An increase in infant deaths in Sedgwick County in 1981 prompted an analysis of the statistics by the Health Department and UKSM-W based on information from birth and death certification. Preterm delivery was the primary cause of infant death. The low fetal death rate coupled with the high rate of first-day deaths suggests obstetric efforts to deliver and resuscitate potential stillbirths.

Infant mortality rate is the ratio of the number of infant deaths to the total number of live births. Similarly, *fetal death rate* is the ratio of the number of fetal deaths to the total number of births (both live and stillbirths).[2]

Method

The study consisted of the resident population of Wichita/Sedgwick County as defined by the mother's "usual place of residency." Requiring Sedgwick County residence as the basis for the study avoided bias which would be introduced if high-risk babies from other counties delivered in Wichita's Level II and III Perinatal Centers were included. Babies born outside of the county to Sedgwick County residents were also included, although these were few in number.

Matched birth and death certificates were obtained for all fetal (41 cases) and infant (97 cases) deaths occurring in 1981 for the defined population. Hospital records were consulted when the certificates were incomplete or if there were questions concerning the accuracy of the information reported.

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TABLE I
INFANT MORTALITY RATES 1977-81

	Wichita/Sedgwick County		Kansas	
Year	*#of Births*	*Infant Mortality*	*# of Births*	*Infant Mortality*
1977	6,138	14.3	36,827	13
1978	6,031	12.6	36,581	12
1979	6,735	12.5	39,916	11
1980	7,094	11	40,686	10.1
1981	7,378	13.2	41,202	11

(Rates per 1,000 live births)

TABLE II
BIRTH RELATED MORTALITY — 1981

	Wichita/ Sedgwick	*Rest of State*	*U.S.A.*
Fetal death*	5.5	8.8	
1-6 days	8.8	5.5	8.9
7-28 days	1.4	1.1	
28-364 days	3.0	3.3	4.3
Infant mortality (1-364)	13.2	9.8	13.1

*Fetal death rate per 1,000 total births. Other rates per 1,000 live births.

Additional statistics were obtained from the Bureau of Maternal and Child Health in Topeka.

Potentially, more than 35 variables could be measured on each baby from the matched birth and death certificates. However, many items (*e.g.*, maternal smoking history) were seldom recorded. A complete comparison of hospital records with birth and death certificates revealed that only 24 items were recorded on the certificates with sufficient accuracy to warrant inclusion in the study. This report focuses on four variables (time of death, birth weight, gestation, and cause of death) to present a brief review of the 1981 fetal and infant mortality data for Sedgwick County. Additional variables will be utilized in future reports.

Results

The large number of births in Wichita/Sedgwick County (18% of all Kansas births) made it necessary to establish a true comparison between Sedgwick County and the rest of the state by calculating a Kansas infant mortality rate that excludes the Wichita/Sedgwick County figures. *Table II* illustrates that the 1981 Sedgwick County infant mortality rate was much higher than that for the rest of the state and that the size of this discrepancy had been partially concealed by including Sedgwick County in the state average. Excluding Wichita/Sedgwick County, the Kansas infant mortality rate was 9.8 per cent, one of the lowest in the United States.[3]

The fetal death rate was markedly lower for Wichita/Sedgwick County than for the rest of the state. One-half of the fetal deaths (51.2%) in Wichita/Sedgwick County were nonmacerated, indicating that delivery took place within a few hours of death. The low fetal death rate coupled with the high rate of first-day deaths suggests obstetric efforts to deliver and resuscitate potential stillbirths. Statistically, this decreases the fetal death rate at the cost of elevating the first-day death rate.

TABLE III
FETAL AND INFANT DEATH BY CAUSE
WICHITA/SEDGWICK COUNTY 1981

	Day of Death					
Principal Cause	*Fetal*	*1*	*2-6*	*7-27*	*28-364*	*Total*
Prematurity	10	37	12	5	0	64
Congenital abnormality	1	7	5	4	5	22
SIDS	0	0	0	1	12	13
Unexplained stillbirth	11	0	0	0	0	11
Placental abruption	10	0	0	0	0	10
Cord accident	7	0	0	0	0	7
Violence/fire	1	0	0	0	5	6
Infection	0	1	2	0	0	3
Hemorrhage	1	1	0	0	0	2
Totals	41	46	19	10	22	138

Time of Death

Examination of the time-of-death variable indicated that the high infant mortality rate in Wichita/Sedgwick County was due to an unusually high number of deaths within the first six days of life (the hebdomadal period). However, in infants older than 28 days, the death rate was slightly lower in Wichita/Sedgwick County than in the rest of the state *(Table II)*.

Within the hebdomadal period, the majority of deaths occurred the first day of life. Indeed, the first day of life accounted for half of the deaths (42/97) in the first year of life for live-born babies. Many of these babies died within minutes of birth and under circumstances that suggest that the classical distinctions between fresh stillbirth and early death in a live-born infant are inappropriate for these babies.

Cause of Death

The primary causes of death for 1981 babies are shown in *Table III*. Prematurity was reported as the principal cause for almost one-half (46%) of all fetal and infant deaths. In fact, prematurity was nearly three times more common than the second most frequently encountered cause of death — congenital malformations.

Congenital heart disease (11 cases with no single diagnosis predominating) was the leading congenital abnormality. The other 11 deaths from congenital abnormalities involved the central nervous system

(five cases), the genitourinary system (three cases) and the pulmonary system (two cases). One case involved severe multiple abnormalities.

Prematurity and congenital abnormality were most influential among babies less than 28 days old. The five remaining deaths, in babies younger than 28 days, were due to infection and hemorrhage. Three cases of infection were disseminated infections contracted in utero: one case each of cytomegalovirus, hepatitis, and staphylococcal infection. Two cases of hemorrhage involved one feto-maternal hemorrhage leading to stillbirth, and an umbilical hemorrhage during an unattended home birth.

Sudden Infant Death Syndrome (SIDS) was the leading cause of death in babies older than 28 days. The 13 SIDS deaths yielded a Sedgwick County rate of 1.76, which is within the expected range of 1.2-3.0/1000 live births.[4]

Trauma — including fire, accident, and assault — was the other important cause of death in older babies. There were no deaths relating to motor vehicle accidents, although one infant was killed in an aircraft crash.

Prematurity, placental abruption, and unexplained intrauterine death were the main causes of fetal death, especially at early gestation. For babies who died after 36 weeks in utero, cord accident was the most frequently stated cause of death.

Birth Weight and Gestational Age

The birth weight distribution of the fetal and infant deaths *(Figure 1)* showed a wide range of birth weights; however, 92 babies (68%) weighed less than 2500g. The distribution of gestational age *(Figure 2)* ranged from 16 to 43 weeks, with 76 babies (58%) less than 36 weeks gestation.

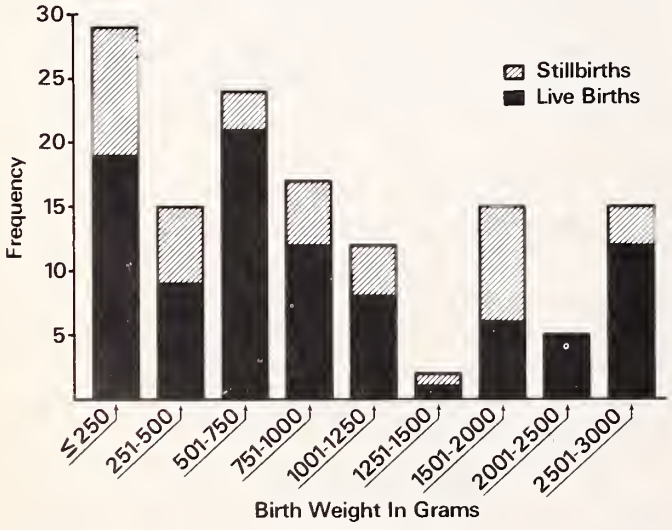


Figure 1. Birthweights for all fetal and infant deaths in 1981.

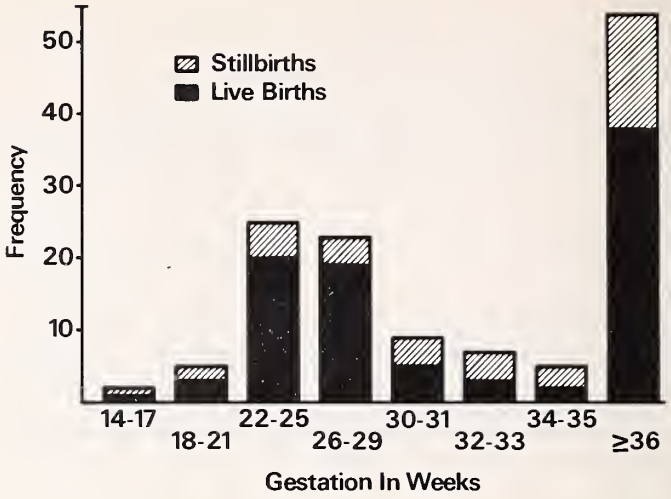


Figure 2. Gestational age for all fetal and infant deaths in 1981.

Since birth weight and gestational age are not independent variables, they were plotted for each baby as coordinates, yielding the cluster diagram shown in *Figure 3*. In interpreting the cluster diagram, traditional measures of prematurity (weight less than 2500g and gestation less than 36 weeks)[5] were used to divide the babies into the following groups:

I. Weight greater than 2500g and gestation greater than or equal to 36 weeks
II. Weight less than or equal to 2500g and gestation less than 36 weeks
III. Weight less than or equal to 2500g and gestation greater than 36 weeks

(There were no babies in the group defined by weight greater than 2500g and gestation less than 36 weeks.) Nine babies were omitted from the cluster diagram since either birth weight or gestation was unavailable.

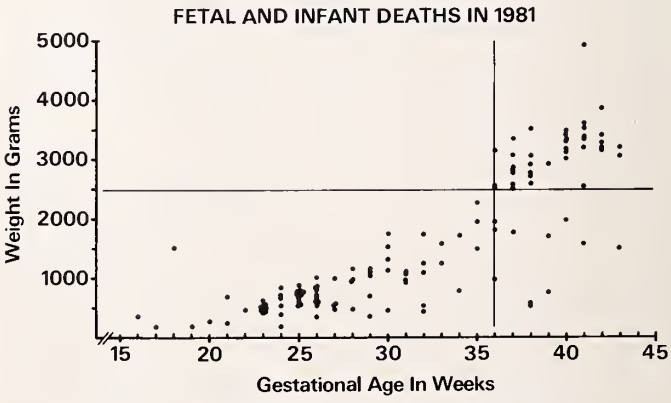


Figure 3. Fetal and infant deaths in 1981.

The 38 babies in Group I could be regarded as mature at the time of death. Their deaths were attributable to congenital abnormalities, SIDS, or violence. Nine babies were late fetal deaths certified as "cord accident" or "unexplained intrauterine death."

Of the 76 babies in Group II who were premature by either weight or gestational criteria, the most frequent cause of death was prematurity (66%), followed by placental abruption (11%), congenital abnormality (8%), intrauterine death (7%), and cord accident (4%). SIDS, infection, and hemorrhage accounted for the remaining Group II deaths.

The 15 Group III babies were light for their gestations. Five babies were multiple births. Three were macerated stillbirths; thus, calculating gestation until the time of birth overestimated the functional growing period. The most frequently listed cause of death for Group III babies was placental insufficiency (seven cases), followed by congenital abnormality (five cases), and infection, hemorrhage, and violence (one case each).

Discussion

This study is based on birth and death certificate documentation, and the results are therefore dependent upon the accuracy of physicians and others who are responsible for these documents. Birth and death certificates are valuable epidemiological sources as well as legal requirements. One of the aims of this article is to raise the awareness of physicians regarding the value of birth and death certificates to provide reliable information on health problems.

Based upon the available death certificate information, prematurity ranks as the dominant cause of fetal and infant mortality in the 1981 Sedgwick County data. Several classical causes of death were noticeable by their absence, such as road traffic accidents. Other expected causes of death occurred in unusual presentations: infection was restricted to three severe acquired infections and was not responsible for the death of any of the older babies. Similarly, the two cases of hemorrhage occurred under very specific circumstances.

Following prematurity, the largest number of deaths were attributed to congenital abnormality (22 cases). Severe cardiac abnormalities accounted for half of these deaths. On the basis of the death certificates, there does not appear to be a specific congenital abnormality that could be targeted as a preventable health problem, such as the alpha-feto-protein screening programs which have been successfully implemented in other countries to detect central nervous system abnormalities. It would be necessary, however, to study survivors as well as fatalities before reaching a firm conclusion on this issue.

The ultimate goal of this retrospective study is to identify areas for prevention of mortality. It is difficult, however, to specify areas where the 1981 deaths could have been prevented given current services and knowledge. This is due either to the severity of the lesions (congenital heart defects), an inability to predict and prevent the deaths (SIDS, unexplained stillbirth, and cord accidents), or human failure (violence or failure to obtain medical help).

The largest number of deaths, however, were attributed to preterm delivery. The usual clinical history indicated that the mother did not present in labor until delivery was inevitable. The problem of late presentation in preterm labor was recently addressed by a San Francisco program.[6] Intensive patient education and counseling by a nurse affiliated with the university hospital obstetric clinic served to reduce the rate of premature deliveries from 6.7 to 2.4 per cent of all births. In addition, the San Francisco program enhanced physician care of each patient by ensuring continuity of care and encouraging compliance with the physician's recommendations. As a result of early detection of preterm labor in a high-risk group of mothers, 93 per cent were suitable for tocolytic therapy on admission, with a mean gain of 6.1 weeks (± 2.6 weeks) in gestation. Such a program offers enormous potential benefit for Sedgwick County in view of the high rate of premature births. The Wichita/Sedgwick County Health Department and the University of Kansas School of Medicine-Wichita are presently seeking support for a local program modeled on the San Francisco study.

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Protamine Anaphylaxis

Protocols for Therapy in Acute Reactions

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PROTAMINE SULFATE is widely used to neutralize the anticoagulant effects of heparin. Rapid intravenous administration of large doses of protamine has been associated with hypotension and bradycardia. Myocardial depression and peripheral vasodilation are allegedly responsible for this hypotensive effect.[1] However, when used properly, protamine is relatively free of severe side effects, although anaphylactic reactions to protamine have been reported. The patient described here developed severe circulatory collapse and bronchospasm following administration of protamine.

Anaphylactic reactions to protamine are infrequent but often lead to severe hypotension and cardiovascular collapse, as in the case of the patient whose successful treatment is described here. Anaphylactic reactions in general, reactions to protamine in particular, and the therapy of these anaphylactic reactions are also discussed.

Case Report

A 59-year-old woman with a 30-year history of insulin-dependent diabetes mellitus was admitted for surgical treatment of severe peripheral vascular disease. Her diabetes was controlled with NPH insulin, 45 units subcutaneously each morning and 10 units subcutaneously each evening. Other medications included nifedipine 10 mg/8 hrs and coumadin 4 mg/day. Past medical history was significant for a three-vessel aortocoronary bypass in 1979 during which she received protamine without any adverse effects. She denied any symptoms of angina pectoris. Significant findings on physical examination were bilateral carotid bruits, decreased pulses in the right lower extremity, and gangrenous changes of the toes of her right foot. Hemoglobin, hematocrit, urinalysis, electrolytes, and blood glucose were all within acceptable limits. The patient was scheduled for bilateral carotid endarterectomy and right femoral-anterior tibial arterial bypass. Each of the three procedures was to be done on separate occasions.

The first surgical procedure was the left carotid endarterectomy. Premedication was meperidine, 20 mg; diazepam, 2.5 mg; and glycopyrrolate, 0.1 mg intravenously. Anesthesia consisted of left superficial and deep cervical plexus block with 40 cc of lidocaine 1.5 per cent with epinephrine 1:400,000. After exposure of the carotid artery, heparin, 14,500 units intravenously, was given prior to crossclamping the internal carotid artery. The patient remained communicative throughout the endarterectomy. Following closure of the arteriotomy, protamine sulfate, 145 mg, was given slowly intravenously during a six-minute period. At this time the patient developed a drop in blood pressure from 180/75 torr to 130/60 torr and complained of substernal chest pain. Heart rate remained unchanged at 100/min. She did not complain of dyspnea or develop bronchospasm. Nitroglycerin 0.4 mg was given sublingually and her chest pain resolved. The surgical incision was closed and she was taken to the surgical intensive care unit without further problems. Blood pressure at that time was 159/65 torr and the pulse rate was 90/min.

Six days later she returned to the operating room for the right carotid endarterectomy. Premedication was meperidine, 20 mg; diazepam, 2.5 mg; and glycopyrrolate, 0.1 mg intravenously. Anesthesia consisted of a right superficial and deep cervical plexus block with 40 cc of lidocaine 1.5 per cent with epinephrine 1:400,000. Heparin, 15,000 units intravenously, was given prior to crossclamping of the right internal carotid artery. Again the patient tolerated crossclamping of the artery and the endarterectomy was performed. Following closure of the arteriotomy, protamine sulfate was given. However, after receiving 10 mg, her blood pressure dropped from 185/60 to 80/30 within several minutes of administration of the drug. She again complained of chest pain. Pulse rate changed from 80/min to 140/min. Phenylephrine, 0.1 mg intravenously, was given immediately as the blood pressure began to drop. Blood pressure rapidly rose to 140/55 torr.

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Propranolol, 0.5 mg intravenously, was given and a dopamine infusion was started. The patient's blood pressure remained stable and her pulse gradually slowed to 100/min during a 20-minute period. The dopamine was discontinued. She was taken to the surgical intensive care unit without further problems.

Five days later she returned to the operating room for the femoral-anterior tibial arterial bypass. Premedication was meperidine, 25 mg; diazepam, 2.5 mg; and atropine, 0.2 mg intravenously. Anesthesia was induced with d-tubocurare, 3 mg; fentanyl, 50 μg; sodium thiopental, 200 mg; diazepam, 7.5 mg; and succinylcholine, 100 mg. This was followed by endotracheal intubation and anesthesia was maintained with nitrous oxide, oxygen, and isoflurane. Heparin, 14,500 units intravenously, was given during the bypass procedure. The entire procedure was unremarkable until protamine, 50 mg, was given intravenously. Within five minutes, the patient's systolic blood pressure declined from 150 torr to 95 torr and pulse declined from 65/min to 45/min. The N_2O and isoflurane were discontinued immediately, and the patient was ventilated with 100 per cent oxygen. Atropine, 0.5 mg, and phenylephrine, 0.2 mg, were given. The lungs became increasingly difficult to ventilate and peripheral cyanosis developed. Blood pressure continued to decline and her pulses became unpalpable. The electrocardiogram showed a sinus bradycardia with a rate of 40/min. External cardiac massage was started. Epinephrine, 1.0 mg (0.5 mg in divided doses); hydrocortisone, 1.0 gm; and sodium bicarbonate, 100 mEq, were given. The bronchospasm cleared and blood pressure and pulse were obtained after these therapeutic maneuvers. Phenylephrine, 0.2 mg, was given and a dopamine infusion was started to support the blood pressure at 110/85 torr. Cimetidine, 200 mg, and diphenhydramine, 25 mg intravenously, were given. Blood pressure stabilized at 170/80 and dopamine was discontinued. Arterial blood gas tensions, pH, and potassium level obtained at this time were PaO_2 127 torr, $PaCO_2$ 41 torr, pH 7.47, and 5.0 mEq/liter respectively. The entire hypotensive episode lasted approximately 15 minutes. The patient was subsequently taken to the surgical intensive care unit and was later extubated without difficulty. She regained full consciousness and had no detectable neurologic deficits.

Discussion

Protamines are simple, low molecular weight proteins found in the semen of fish. They have a large arginine content and are, therefore, strongly basic molecules.[2] Heparin is a strong organic acid with a negative charge that combines with the positively charged protamine to form a neutral salt.

Anaphylactic reactions following the administration of protamines occur unpredictably. There is agreement, however, that patients with a chronic history of atopy are more likely to experience allergic reactions to drugs administered intravenously.[3] This increased susceptibility is primarily a reflection of a genetic predisposition of these patients to form increased amounts of immunoglobulin E (IgE) antibodies. The likelihood of an allergic reaction increases with the number of exposures to a drug.

Anaphylaxis requires previous exposure to the drug or a chemically similar substance and the production of IgE antibodies. These IgE antibodies attach to receptors on mast cells and basophils. Degranulation of these cells and release of chemical mediators result when the patient is re-exposed to the drug. The primary chemical mediator is histamine. Histamine is a potent vasodilator and causes hypotension if released in large enough quantities.

The drug can also have a direct effect on mast cells and basophils that stimulates release of histamine. This is called an anaphylactoid reaction. The manifestations of an anaphylactoid reaction are indistinguishable from those of an anaphylactic reaction.

Once diagnosed, an anaphylactic reaction must be treated promptly. The initial steps should include discontinuing administration of the allergen, ventilation with 100 per cent oxygen, and discontinuation of anesthetic agents. Rapid restoration of adequate circulatory volume is necessary. This should be done with crystalloid, colloid, or blood. The large decrease in circulatory volume results from a loss of fluid into the interstitial spaces.[8]

Drug therapy of an anaphylactic reaction consists of administering epinephrine, antihistamine, and aminophylline.[3] Epinephrine, in doses of 0.5 to 1.0 mg intravenously, is the mainstay of treatment. Epinephrine is a potent vasoconstrictor and restores blood pressure and therefore coronary and cerebral perfusion pressures in hypotensive patients. An infusion may also be used to maintain blood pressure once it is restored. Epinephrine is also a potent bronchodilator and may be effective treatment for the bronchospasm that often occurs during a reaction. If the blood pressure is restored by the epinephrine and the bronchospasm persists, aminophylline should be given. The loading dose for aminophylline is 7 to 9 mg/kg during a 20 minute period. Antihistamines should be given to help diminish the effects of the histamine released. The antihistamines generally used are diphenhydramine

— an H_1 antagonist — and cimetidine — an H_2 antagonist.

Other drugs that may be helpful in the treatment of anaphylactic reactions are isoproterenol and corticosteroids. Bronchospasm that persists after treatment with epinephrine and aminophylline can be treated with isoproterenol. Isoproterenol is a potent bronchodilator. It must be given with caution to hypotensive or hypovolemic patients. It may cause vasodilation by beta-adrenergic stimulation and aggrevate the hypotension. Corticosteroids have not been proven to have a beneficial effect in acute reactions, but are frequently given and may be useful.

Anaphylactic reactions to protamine have been reported in several different situations. Several case reports in diabetics receiving insulin containing protamine have been reported. Moorthy *et al.*[4] reported an anaphylactic reaction to 20 mg of protamine in a young diabetic woman whose diabetes was controlled with protamine zinc insulin. Vontz *et al.*[5] reported an anaphylactic reaction to 10 mg of protamine in a diabetic male whose diabetes was controlled with NPH insulin. The only exposure either of these patients had to protamine prior to their anaphylactic reactions was to the small amounts of protamine in their insulin preparations.

The patient reported here was also an insulin-dependent diabetic and had been receiving NPH insulin for many years. However, she had been receiving this insulin prior to her aortocoronary bypass surgery in 1979 and did not have an allergic reaction to the protamine she received at that time.

Fish allergy has also been associated with anaphylactic reactions to protamine. Knape *et al.*[6] reported a severe anaphylactic reaction to protamine following aortocoronary bypass surgery in a patient who was allergic to fish. Coplan and Berkman[7] reported four patients who had allergic reactions to protamine they received following blood component donation. Two of these patients were found to be allergic to fish. A relationship between fish sensitivity and protamine sensitivity is conceivable when the derivation of protamine from salmon sperm is considered.

The patient described appears to have experienced an anaphylactic reaction to protamine on each of her last two exposures to the drug. Her mild hypotensive response during the left carotid endarterectomy was more consistent with the effects normally seen with routine use of protamine. Her reaction during the right carotid endarterectomy was consistent with a mild anaphylactic reaction and was easily treated with a vasopressor and propranolol. This reaction was probably mild due to the small dose of protamine administered. Her reaction during the last surgical procedure was a severe anaphylactic reaction with hypotension, bronchospasm, and bradycardia. This was adequately treated with epinephrine, antihistamines, and steroids. The bronchospasm cleared with the use of epinephrine; therefore aminophylline was not necessary.

In summary, this patient apparently had several anaphylactic reactions to protamine. Sensitization might have occurred either by chronic administration of NPH insulin or by exposure to the drug on several occasions during her previous surgical procedures. Anesthesiologists should exercise caution when administering protamine to patients with insulin-dependent diabetes mellitus or a history of allergy to fish. These groups of people appear to have an increased incidence of anaphylactic reactions to protamine.

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Hemorrhage During Myringotomy

Probable Causes and Appropriate Treatment

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Myringotomy is a common surgical procedure usually requiring general anesthesia. An uncommon complication is hemorrhage. Presented is a case of significant blood loss during surgery. Since this complication is seldom reported in the anesthesia literature, this complication is reported with discussion of the probable causes of hemorrhage during myringotomy.

Case Report

A 21 month 12.8 kgm white male was admitted to the one-day-care unit with a one month history of bilateral otitis media. At the time of admission, he was afebrile; there was no drainage from the ears; and he was taking antibiotics. Other than the ear lesion, the history and physical examination revealed nothing unusual.

The child was brought to surgery unpremedicated. After placement of EKG, chest stethoscope, and blood pressure cuff, anesthesia was induced and maintained with halothane, N_2O-O_2, by mask. No intravenous infusion was started.

As the surgeon was making an incision in the tympanic membrane, there was sudden bleeding (estimated blood loss 125 ml). The bleeding was controlled with cotton packed tightly in the external auditory canal. An intravenous infusion of 500 ml of 5 per cent Ringers lactate was started, and the child was crossmatched and subsequently given blood. After 30 minutes the packing was removed; the bleeding had stopped. No other manipulation was attempted and the ear canal was repacked. Myringotomy was then performed on the opposite ear without incident. The child recovered uneventfully.

Discussion

The probable causes of hemorrhage during myringotomy relate to the anatomy of the vascular structures and their nearness to the tympanic membrane.

Normally the superior jugular bulb is formed by the joining of the sigmoid and inferior petrosal sinuses which then continue as the internal jugular vein. The bulb lies in the jugular fossa which is located below the floor of the tympanic cavity. Normally the fossa is separated from the cavity by a thin sheet of bone.

Myringotomy is a common surgical procedure. Although hemorrhage is an uncommon complication, advance understanding of the probable causes and appropriate treatment will facilitate a quick and effective response should it occur.

The internal carotid artery enters the petrous bone in the carotid canal. It ascends vertically anterior to the tympanic cavity, then turns sharply anteromedially to pass inferiorly to the Eustacian tube. Where the internal carotid lies anterior to the tympanic cavity, it is separated from the cavity by the thin tympanic plate which forms the posterior wall of the carotid canal and the anterior wall of the tympanic cavity.[1]

The most common vascular abnormality is a high jugular bulb. The incidence of a jugular bulb projecting above the tympanic annulus into the middle ear is 6 per cent. There may be absence of the bony plate separating the jugular fossa from the tympanic cavity which may allow the jugular bulb to impinge on the middle ear. The incidence of this finding is 7 per cent.[2] Even if the bony covering is present with a high bulb, it is thin and may easily be perforated.

A more uncommon anomaly is an aberrant carotid artery that enters the tympanic cavity.[3, 4] In the last 15 years there have been several reports of this aberration.[2-5] The incidence is 1 per cent.[4]

There have been a few instances of the rarer anomaly, aneurysm of the internal carotid artery projecting into the tympanic cavity.[6, 7]

The difficulty in assessing anomalies of this nature is that they may coexist with otitis media or they may mimic otitis media. They may appear as a reddish or bluish discoloration behind the tympanium.[7] They may produce a variety of symp-

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toms ranging from fullness, earache, tinnitus, vertigo, and irritation of the tympanic membrane. They can be mistaken for glomus tumors of the middle ear.

Differentiation of these abnormalities are made radiographically. They may be demonstrated by tomography or contrast studies. Despite the validity of radiographic studies, the aberrations are subtle, requiring a high index of suspicion. A high jugular bulb can be seen with retrograde jugular venography. An anterior-posterior arteriogram will demonstrate the aberrant internal carotid as passing lateral to a line drawn vertically down from the vestibule. In a study of 100 normal subjects, the internal carotid consistently presented medial to the vestibular line.[8]

Of these abnormalities, the high jugular bulb, while the most common, is also the most easily controlled in the event of bleeding from inadvertent puncture. Hemorrhage can usually be controlled with Gelfoam and pressure without severe blood loss. In the event of the less common arterial location, the blood loss may be quite severe. In addition it may be very difficult to control the hemorrhage with pressure alone. Vascular surgery may be required either to ligate the internal artery in the neck or occlude the internal carotid with a balloon catheter.[5]

The anesthesiologist must be prepared to support vascular volume until the hemorrhage can be controlled. This requires an intravenous access to provide fluid support. It may also be prudent to crossmatch blood where middle ear anomalies are suspicioned by x-ray or surgery is anticipated for a glomus tumor.

Summary

A case of sudden hemorrhage during myringotomy in a child is presented. Awareness and fundamental preparations should be immediately available for handling this complication.

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Riley-Day Syndrome

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Mannitol Intoxication

Physiologic Consequences of Overdose

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MANNITOL is the reduced form of the sugar mannose, and is widely employed diagnostically and therapeutically in medical practice. Mannitol is a hexahydric alcohol with the composition $C_6H_8(OH)_6$. Mannitol's usefulness depends upon its ability to induce a non-electrolyte solute diuresis.

Intravenously infused mannitol does not cross cell membranes, and mobilizes intracellular water into the extracellular space. Mannitol is an inert substance and exerts no biologic or metabolic effects in the body. Its sole effect is osmotic. Eighty per cent of infused mannitol is excreted in the urine after glomerular filtration. There is little renal tubular modification of mannitol.

An infusion of mannitol results in an increase in extracellular volume and plasma volume with a concomitant decrease in blood viscosity. Because mannitol is so easily filtered by the kidneys, the increase in plasma volume is not significant. In the presence of renal disease with decreased glomerular filtration, excessive mannitol infusion may precipitate pulmonary edema. There is, however, a large margin of safety associated with the use of mannitol and the agent has wide application.

Mannitol is widely used for prophylaxis and treatment of acute renal failure. Mannitol is effective in aborting incipient renal failure by increasing cardiac output and renal blood flow, by decreasing blood viscosity in the renal microcirculation, and by establishing an increased volume of urine which prevents tubular obstruction with sludge.[1] Non-renal application of mannitol makes use of its ability to induce fluid shifts across the cell membrane. Mannitol has been used to reduce elevated intracranial pressure by mobilization of cerebral edema fluid. Mannitol is employed in the treatment of glaucoma, respiratory distress syndrome, and symptomatic dialysis dysequilibrium.

Complications of mannitol therapy are rare despite its widespread use in medical practice. The few case reports of mannitol intoxication in the literature describe it, for the most part, in patients with preexisting acute or chronic renal failure.

Reported here is the case history of a patient who developed acute renal failure following a massive, accidental overdose of mannitol. The evolution of clinical findings in this patient presented a unique opportunity to observe the physiologic effects of mannitol.

Mannitol, an inert substance that exerts no biologic or metabolic effects in the body, is used, with few adverse reactions, for a variety of diagnostic and therapeutic procedures. In the case presented here, an accidental massive overdose presented a unique opportunity to observe the physiologic effects. Hemodialysis is the most efficient treatment for mannitol overdose.

Case Report

A 39-year-old previously healthy male was hospitalized with a four-hour history of abrupt onset of colicky right flank pain associated with vomiting. A renal calculus was suspected to be present and intravenous pyelography did demonstrate an opaque calculus impacted in the mid right ureter. An intravenous infusion of 5 per cent dextrose in water was begun at 125 ml/hr and subsequently changed to 5 per cent dextrose in one-half normal saline. The attending physician ordered an infusion of one 500 ml bottle of 20 per cent mannitol daily in an attempt to dislodge the stone by increased urinary flow created by the osmotic diuresis of mannitol. Nursing personnel misunderstood the order and instead of one bottle being administered daily, a continuous infusion of 20 per cent mannitol was established at a rate of 170 ml/hr. During a period of 60 hours, the patient received a total 1100 gm of intravenous mannitol. The dosage range normally is 12.5-200 gm daily in healthy adults. At the time of discovery of the infusion error the patient was found to have a blood pressure of 180/115 mmHg and a pulse of 90/minute. An S-4 gallop was audible at the apex of the heart. The cardiovascular physical findings reflected the increased plasma volume expected from mannitol infusion. Despite these findings, however,

the patient's appearance was one of dehydration with dry skin and mucous membranes.

The pre-eminent bedside findings were mental confusion and lethargy due to cerebral desiccation. Nausea and vomiting were present, and had progressively worsened as body water was lost through mannitol diuresis.

The patient's obtundation was significant enough to result in hypoventilation as can be inferred from blood gas studies drawn at the time of discovery of the mannitol error. At this time the pCO_2 was 60 mmHg and a mild respiratory acidosis was present with a pH of 7.26. The pO_2 was 110 mmHg on low flow supplemental nasal oxygen. Mannitol infusion dilutes extracellular bicarbonate concentration with mobilized intracellular water and this may result in a metabolic acidosis. A metabolic component to the patient's acidosis was not present, and serum bicarbonate remained at 26 mEq/L on several determinations.

As the osmotic activity of mannitol causes water to move from the intracellular to the extracellular space, it likewise results in a decrease in extracellular sodium and chloride concentrations. The patient's serum sodium reached a low of 106 mEq/L. If an osmotic diuresis is established with mannitol the loss of large amounts of hypotonic urine may result in hypernatremia; it did not happen in this patient because supplemental hypotonic fluids were being given. He also developed oliguria following the diuresis induced by the excessive mannitol. These two conditions resulted in hyponatremia rather than hypernatremia in this instance. Serum chloride paralleled the fall in serum sodium and was proportionately as low as the serum sodium.

Mannitol infusion experimentally in both animals and humans usually results in hyperkalemia, especially in a setting of pre-existing renal failure and a resultant impaired ability to excrete potassium.[2] The patient was found to have a serum creatinine of 1.8 mg/dl on admission laboratory studies, and this value indicated mild renal failure associated with the obstructing renal calculus. During mannitol infusion the serum potassium rose as a result of transcellular migration of potassium due to solvent drag in association with the mobilization of intracellular water. As progressive dehydration and renal insufficiency supervened, and impaired renal potassium excretion, the serum potassium rose to a high of 6.3 mEq/L. Renal function studies following the mannitol infusion indicated the presence of renal failure with a serum creatinine of 6.2 mg/dl and blood urea nitrogen of 33 mg/dl. The low value for blood urea nitrogen relative to creatinine reflects dilution of extracellular fluid and perhaps wash out of urea during the osmotic diuresis. The literature includes no case reports of mannitol being intrinsically toxic to the kidneys; therefore it appears that the oliguric renal failure seen in this patient was a result of the osmotic diuresis induced by mannitol. This conclusion is supported by the rapid recovery in renal function and re-establishment of normal urine flow seen after removal of mannitol with hemodialysis and fluid replacement. Renal functional parameters obtained at the time of discovery of the infusion error pointed to a mixed pre-renal and renal component to the oliguric renal faiilure.

Serum osmolality measured after the mannitol infusion was 358 mOsm/kg. The serum osmolality was also calculated according to the following formula:

Serum osmolality (mOsm/kg) = (2 × Serum sodium concentration) + Urea osmolality + Glucose osmolality

Urea osmolality is determined by dividing the blood urea nitrogen concentration by 2.8. The glucose osmolality is determined by dividing the serum glucose by 18. The following values were obtained after the mannitol infusion and were used in the equation to calculate serum osmolality: Serum sodium, 106 mEq/L; blood urea nitrogen, 33 mg/dl; and blood glucose, 139 mg/dl. The serum osmolality was calculated to be 232 mOsm/kg compared to the measured value of 358 mOsm/kg. This serum osmolar gap of 126 mOsm/kg represents mannitol.

Mannitol infusion ordinarily lowers the hematocrit and hemoglobin values but just the opposite was found to be present in this patient. Following mannitol infusion the hemoglobin was 16.7 gm/dl and the hematocrit 51%. This paradoxical hemoconcentration can be ascribed to a loss of extracellular fluid induced by the osmotic diuresis.

The infusion of mannitol resulted in progressive intracellular desiccation as water was osmotically shifted to extracellular compartments. With establishment of a profound osmotic diuresis, progressive loss of extracellular water ensued leading to oliguric renal failure. It is conceivable that the serum sodium could have fallen further precipitating seizures had the patient not developed oliguria.

Treatment options in the patient included replacement of water and electrolytes to re-establish urine flow and permit excretion of the infused mannitol, or removal of the mannitol with either hemodialysis or peritoneal dialysis.

Fluid administration in this oliguria patient would

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Erectile Dysfunction

A Comprehensive Approach to Evaluation

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PENILE TUMESCENCE is a complex process involving coordination of neurological, vascular, hormonal, and psychological systems. Erectile failure may result from dysfunction in any one or several of these. With the development of new treatment modalities and current evidence that physical disorders and medications may be primary causative factors in 50-60 per cent of patients with erectile dysfunction, systematic assessment of patients to clarify etiology is extremely important.[1] The development of several new technical procedures, discussed below, has facilitated more accurate and specific diagnoses, allowing the clinician to select the most appropriate treatment modality. The approach of the UKSM-Wichita Sexual Medicine Clinic to the evaluation of patients with erectile dysfunction is outlined. The case report illustrates the need for thorough evaluation with each patient, even in the presence of an obvious presumptive etiology.

Penile tumescence is a complex process involving coordination of several systems. Contrary to earlier beliefs, it is now known that fewer than one-half of erectile dysfunctions are psychogenic. Comprehensive evaluation with current diagnostic procedures and treatment methods can effect a satisfactory resolution of the problem for many patients.

Case Report

A 58-year-old married white male with a 13-year history of bipolar affective disorder was referred by his psychiatrist for evaluation of erectile dysfunction. His chief complaint was "I can't have an erection." He reported isolated episodes of erectile failure at approximately ages 32 and 39 when under significant stress at work. These transient episodes resolved with reduction in job stress, and he maintained a healthy sexual relationship with his wife, with intercourse two or three times/week, until approximately 1971, at which time he was begun on tricyclic antidepressants following his first major depressive episode. He described a progressive decline in erectile capacity, manifested first by an inability to maintain the quality of erections, and then by inability to obtain in any setting (intercourse, masturbation, or with erotic stimuli) erections of sufficient rigidity to allow penetration. For approximately 1.5 years prior to admission, he was not aware of having any nocturnal or morning erections, except occasionally upon awakening with a full bladder, and this erection detumesced after voiding. There was no associated decline in sexual desire until the past several months. Ejaculatory function was preserved, except for diminished sensation. Semen was of normal color, and he reported no pain with ejaculation. He indicated that his inability to achieve erections had placed a "strain" on the marital relationship since his wife did not enjoy manual stimulation. As a result, they have ceased attempting sexual relations.

Past medical history was significant for the following: 1963: vasectomy without apparent psychological sequelae, and hemorroidectomy twice; 1967: severe prostatic infection with bloody and painful ejaculation; 1971 and 1981: hospitalizations for major depressive episodes; 1982: hospitalization for recurrent diarrhea.

Medications at the time of evaluation were imipramine, 200 mg/day and Stelazine, 2 mg two times/day (although the patient had a history of adjusting his own medications). Habits: He has an 80 pack/year history of cigarette smoking, denied significant alcohol use, and drinks 6-8 cups of coffee daily.

Family history was positive for bladder cancer in father, a positive history of hypertension, stroke, and myocardial infarction.

Review of systems was positive for brief "blackout" when tilting head backwards (*i.e.*, to look upwards) but without actual loss of consciousness; cold lower extremities; numbness and tingling of lower extremities approximately two times/week;

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diminished fine motor coordination; decreased libido for two to three months; increased intolerance of cold weather; hesitancy, frequency, and nocturia.

Mental Status

The patient was an alert, fully oriented, mildly anxious, moderately obese white male who appeared older than his stated age. Gait was somewhat clumsy, but there was no psychomotor retardation. Behavior was entirely appropriate during the interview. Speech was somewhat hesitant but without thought disorder. Mood was described as "about normal" and assessed as euthymic without evidence of depressed quality. There were no hallucinations or delusions, and cognition was grossly intact.

Physical Examination

Blood pressure was 146/95 mmHg; pulse, 65/min; respirations, 14/min. Funduscopic examination revealed "copper-wiring" and 2^+ AV nicking; the remainder was within normal limits. Neck and cardiac examination were within normal limits and no bruits were auscultated. Chest examination revealed decreased breath sounds at bases, hyperresonance on percussion, and mild increase in AP diameter. There was no gynecomastia. Abdomen was moderately obese; liver span was 14 cm by percussion without nodularity or tenderness. There were no abdominal or femoral bruits. Femoral pulses were $2^+/4$, dorsalis pedis and posterior tibial pulses were trace bilaterally. Toes were mildly cyanotic, and feet were cool to touch bilaterally. Genital examination was normal except for left varicocele, numerous cherry angiomata over the scrotum, absent bulbocavernosus reflex. Cremasteric reflex was intact. Rectal examination revealed normal sphincter tone and perianal sensation but absent anal wink reflex. Prostate was asymmetrically hypertrophied, with left lobe markedly larger than the right, but no nodularity. Neurologic examination was normal except for mild ataxia, diminished fine motor coordination, decreased balance on Romberg, diminished vibratory sensation in all four extremities, and absent Achilles tendon reflex bilaterally.

Screening Laboratory Studies

Results of laboratory tests were: CBC, normal; multi-chem, normal except for cholesterol 266 mg/dl, alkaline phosphatase 164 μ/l; thyroid profile, normal; testosterone (AM), 480 ng/dl (lab nl 300-1200); serum B_{12} and folate, normal; imipramine level, therapeutic.

Formulation and Recommendations

The diagnosis was erectile dysfunction (DSM III: Inhibited Sexual Excitement), thought to be secondary to vascular disease and possibly medication side effects. Since the outpatient evaluation revealed several additional abnormalities, the patient was admitted to Wesley Medical Center for further diagnostic workup and for nocturnal penile tumescence studies in the Wesley Neurodiagnostic Laboratory.

Doppler ultrasonography revealed that the dorsal artery pulse of the penis was faint and difficult to detect, but arterial flow was demonstrated at the base of the shaft. These findings were consistent with extremely low pressure in the dorsal artery of the penis.

Digital angiogram of the head and neck were within normal limits except for minimal narrowing at the origin of the left vertebrae.

Nocturnal penile tumescence (NPT) studies showed moderate tumescence in association with REM periods, but with insufficient rigidity. There was diminished total NPT time for the patient's age, but this was felt to be related to the reduction in total amount and percentage of REM and increase in REM latency (effects seen both in depression and with imipramine). The findings of partial tumescence with diminished rigidity is consistent with a vascular etiology of the erectile dysfunction.

Urologic consultation was obtained and cystoscopy revealed multiple tumors on the posterior bladder wall. These were subsequently resected and found to be Grade I, non-invasive, transitional cell carcinomas. Resection of the prostate was to be considered at a later date. This patient may be considered for a penile prosthesis pending further therapeutic decisions regarding his prostatic hypertrophy and bladder carcinoma.

Discussion

This patient illustrates the importance of a systematic, comprehensive evaluation of patients with erectile dysfunction. Although the obvious "causes" in his case might have appeared psychological (depression, marital difficulties) or medication effect, several additional and highly significant factors were identified in the course of his assessment. The traditional bias of the medical profession and lay public has been that erectile failure is psychogenic in 95 per cent of patients. Improved diagnostic capability, however, has shown that the problem has a primary organic etiology in at least

one-half of the cases. In one series of 120 patients, Gray[2] and co-workers determined the following etiologic diagnoses: vasculogenic — 45; diabetes mellitus — 24; psychogenic — 14; Peyronie's disease/priapism — 14; neurogenic — 8; testosterone deficiency — 4; malignancy — 7; and trauma — 4. Thus, erectile failure is a multifactional disorder with numerous medical, surgical, and pharmacologic etiologies now appreciated. These are summarized in *Table I*.

A review of the normal physiology of penile tumescence is helpful to understand the mechanisms by which normal function may be impaired. Erection is a hemodynamic event, mediated via the autonomic and somatic nervous systems. Penile blood supply is from terminal branches of the internal pudendal arteries: the dorsal artery of the penis; deep penile arteries supplying the corpora cavernosa; the branch to the corpus spongiosum; and perineal, scrotal, and urethral arteries. In response to visual, auditory, olfactory, gustatory, tactile, proprioceptive, and cortical stimuli, arteriolar dilation and shunting of blood into the corporal sinuses occurs, causing tumescence. There are thought to be two spinal centers mediating erectile function: the thoracolumbar center (T12-L1) regulating erections occurring in response to cortical stimuli *(psychogenic erections)* via the sympathetic outflow and the hypogastric plexus; and the sacral center (S2, S3, S4) thought to mediate erections produced by direct sensory stimulation of the penis *(reflexogenic erections)*. Sacral innervation involves somatic, parasympathetic, and sympathetic fibers. Somatic sensory impulses are transmitted to the sacral cord via the pudendal nerves (S2-S4). Efferent impulses travel from the sacral cord in the parasympathetic fibers (''nervi erigentes'') to the penile vasculature. Short adrenergic nerves in the corpora cavernosa are also involved. Erection was considered a cholinergic event, but current evidence is that the final common pathway is via the neurotransmitter norepinephrine acting on beta-adrenergic receptors in arterioles of the corpora.[1]

TABLE I
MEDICAL DISORDERS ASSOCIATED WITH ERECTILE DYSFUNCTION

Endocrine	Pituitary disorders (acromegaly, chromophobe adenoma, cranio-pharyngioma, pituitary ablation, hyperprolactinemia), Addison's or Cushing's syndrome, hyperthyroidism (thyrotoxicosis), hypothyroidism (myxedema), gonadal dysfunction (castration, post-inflammatory fibrosis; exogenous estrogens; Klinefelter or male Turner syndromes; feminizing interstitial-cell tumor) pancreatic disorders (diabetes), Fröhlich's syndrome
Neurologic	Myasthenia gravis, multiple sclerosis, Parkinsonism, amyotrophic lateral sclerosis, stroke, cerebral tumors, infections (particularly lesions of temporal lobe), head trauma, spinal cord trauma, spinal cord compression (disc, tumor, or abscess), tabes dorsalis, temporal lobe epilepsy, spina bifida, syringomyelia, subacute combined degeneration of the cord, peripheral neuropathy, cerebral palsy
Vascular	Large vessel disease (atherosclerosis), small vessel disease (arteritis), thrombosis (particularly of aortic bifurcation), embolism, aneurism, Leriche's syndrome
Pharmacologic	Multiple pharmacological agents affect sexual function; these are summarized in *Table II*.
Postoperative	Perineal prostatic biopsy, perineal prostatectomy (frequently), suprapubic and transurethral prostatectomy (occasionally), abdominal aortic aneurysmectomy, aortofemoral bypass, retroperitoneal lymphadenectomy, sympathectomy (lumbar, dorsal, pelvic), cystectomy, abdominoperineal resection, external sphincterotomy
Endurance-related	Myocardial failure, angina pectoris related insufficiency, anemia, leukemia, systemic illness (infections, nutritional deficiencies, immunologic disorders), metabolic diseases (renal or hepatic failure), sickle cell disease
Mechanical	Congenital deformities (extrophy, epispadias, hypospadias, chordee, microphallus), Peyronie's disease, morbid obesity, hydrocele, spermatocele, varicocele, phimosis, priapism, urethral stricture
Inflammatory	Urethritis, prostatitis, seminal vesiculitis, cystitis, gonorrhea, tuberculosis, elephantiasis, mumps
Traumatic	Penectomy (surgical or traumatic), urethral rupture, pelvic fracture

TABLE II
DRUGS ASSOCIATED WITH SEXUAL DYSFUNCTION*

Drug Class	*Phase of Sexual Response Cycle Affected*	*Hypothesized Mechanism of Effect*
Anxiolytics	Decreased libido; ejaculation difficulties	CNS depressant effect on limbic system
Anticholinergics	Erectile dysfunction	Inhibit acetylcholine at postganglionic nerve ending, thereby inhibiting parasympathetic nervous system, resulting in impairment of reflex vasocongestion of penis needed for tumescence
Antidepressants		
Tricyclics	Decreased libido; erectile dysfunction; ejaculation difficulties	Sedating properties (histamine H1 receptors) result in decreased libido. Anticholingeric activity results in erectile dysfunction. Ejaculatory difficulties due to alpha adrenergic blockade
MAO Inhibitors	Erectile dysfunction; ejaculation difficulties	Via autonomic effects
Antihistamines	Decreased libido; erectile difficulties	Cimetidine may produce hormone alteration. All cause decreased libido due to sedating effects; decreased erectile capacity via antagonism of acetylcholine centrally and peripherally
Antihypertensives		
Diuretics		
Spironolactone	Decreased libido; erectile dysfunction	May produce decreased erectile function in 30% of patients; steroid analog whose hormonal disturbances may be due to antiandrogen activity at tissue receptor
Thiazides Furosemide Ethacrynicacid	Erectile dysfunction; ejaculation difficulties	? due to blunting of vascular reflexes via influence on smooth muscle reactivity. ? due to hyperglycemic and hypokalemic effects. Incidence: may reach >30%
Centrally Acting Sympatholytics		
Alpha methyldopa	Decreased libido; erectile dysfunction; ejaculation difficulties	Dose-related incidence up to 50% of patients when dose >2.0 g/day. Effects mediated by central nervous system (CNS) catecholamine depletion and production of false neurotransmitter
Clonidine	Decreased libido; erectile dysfunction; ejaculation difficulties	Incidence 10-20%. Effects may be mediated by alpha adrenergic blocking activity (centrally and peripherally)
Reserpine	Decreased libido; erectile dysfunction; ejaculation difficulties	Depletes CNS catecholamines producing sedation causing decreased libido; also causes increased prolactin resulting in gynecomastia
Alpha Adrenergic Blockers		
Phentolamine	Erectile dysfunction ?; ejaculation difficulties	Due to alpha adrenergic blockade resulting in inhibition of emission and ejaculation
Phenoxybenzamine	Erectile dysfunction ?; ejaculation difficulties	

(Continued on next page)

Drug Class	*Phase of Sexual Response Cycle Affected*	*Hypothesized Mechanism of Effect*
Beta Adrenergic Blockers		
Propanolol	Decreased libido; erectile dysfunction	Sexual side effects possible but less common. Low incidence of decreased libido (1-3%). Reported
Metoprolol	Erectile dysfunction ?	case of Peyronie's disease with metoprolol
Ganglion Blockers		
Mecamylamine	Erectile dysfunction; ejaculation difficulties	Rarely used in U. S. due to paralysis of peripheral autonomic nervous system leading
Pentolinium	Erectile dysfunction; ejaculation difficulties	to loss of entire sexual response. Trimethaphan used for hypertensive crises
Trimethaphan	Erectile dysfunction; ejaculation difficulties	
Sympathetic Nerve Terminal Agents		
Guanethidine	Decreased libido?; erectile dysfunction; ejaculation difficulties	Deplete norephinephrine stores and prevents its release, causing either failure of ejaculation or retrograde ejaculation in as many as 60% of patients; impotence reported in 30%
Non-Adrenergic Vasodilators		
Hydralazine	Decreased libido (at high doses); erectile dysfunction (at high doses)	Direct relaxation of arteriolar smooth muscle; relatively free of sexual side effects except at doses >200 mg/day, when 5-10% of men report decreased libido, occasionally accompanied by erectile dysfunction
Prazosin	Decreased libido; erectile dysfunction (rare)	Blocks vascular post-synaptic alpha adrenergic receptors causing vasodilation. Decreased libido in 15% of patients
Antipsychotics		
Phenothiazines	Decreased libido; erectile dysfunction; ejaculation difficulties	Effects mediated via potent adrenergic blocking activity (ejaculatory difficulties), anticholinergic activity (erectile dysfunction) and endocrine effects due to action on hypothalamus
Butyrophenones	Decreased libido ?; erectile dysfunction	Incidence of impotence estimated 10%, perhaps related to hyperprolactinemia as a result of dopamine blockade in CNS; galactorrhea and gynecomastia are common side effects
Narcotics	Decreased libido; erectile dysfunction; ejaculation difficulties	General CNS depression and possible direct depression of CNS sex centers; alteration of normal CNS balance of biogenic amines; decreased serum testosterone via active endocrine effect
Miscellaneous		
Clofibrate	Decreased libido; erectile dysfunction	Mechanism unknown
Digitalis	Decreased libido?; erectile dysfunction	Estrogen-like effects (chemical similarity) decreased serum testosterone
Disulfiram (Antabuse)	Erectile dysfunction; ejaculation difficulties	Mechanism unclear (? drug effect or result of chronic alcoholism)
Lithium	Erectile dysfunction (may be possible)	Due to endocrine changes (hypothyroidism, decreased serum testosterone, etc.)
Marijuana	Decreased libido; erectile dysfunction	Decreased serum testosterone and other endocrine effects
Tryptophan	Decreased libido; erectile dysfunction	Increased CNS concentration of serotonin

* Adapted from reference 1.

Current research suggests that erectile function may be suppressed by increases in central serotonin levels, and augmented by elevations of dopamine. For example, haloperidol, a dopamine receptor antagonist, has been associated with production of impotence in patients, while the dopaminergic agonist, L-Dopa, has been shown to increase sexual activity in patients with Parkinson's disease.[1] Erection is not an "all-or-none" phenomenon. Vascular insufficiency may result in erections that are inadequate for intercourse either because they are too soft to allow intromission (insufficient arterial flow and pressure), or are of such brief duration that completion of intercourse is not possible. Gray[2] regards this phenomenon as analogous to claudication in which a high demand for blood flow cannot be met. Ginestie and Romieu[3] have shown that normal erectile function is not possible with only one intact internal pudendal axis; however, additional research is needed to define precisely the lower limits of vascular patency that will allow normal erections of sufficient rigidity for intercourse.

The presence of two spinal centers governing erection has important clinical implications. Patients with complete cord transections above the sacral center but below the thoracic center may still be capable of *psychogenic* erections mediated by impulses descending from cortical and subcortical centers and exiting the cord at T12-L1 outflow. With a cord lesion above both spinal centers, psychogenically induced erections are blocked, but the patient may still be capable of *reflexogenic* erections from direct tactile stimulation of the penis even though he is unable to experience the sensation.[4]

Numerous pharmacologic agents may affect any phase of the sexual response cycle and these are summarized in *Table II*. The particular effect in a given individual will vary, depending upon such factors as age, weight, dosage, absorption, rate of metabolism and excretion, duration of use, presence of other drugs, patient compliance, and suggestibility.[5] The incidence of adverse effects of drugs on sexual function is difficult to quantify. Hogan[6] studied 861 male patients receiving antihypertensive medication, compared with an age-matched control group of 177 normotensive males on no medication. He reported incidences of overall sexual dysfunction (not just limited to erectile dysfunction), ranging from 6-40 per cent for propanolol, 2-26 per cent for methyldopa, and 1-20 per cent for clonidine. Continued research is needed to clarify the magnitude of sexual side effects of frequently prescribed medications. Knowledge of the potential sexual side effects of various therapeutic (as well as illicit or "recreational") drugs is important to allow selection of alternative medication should an adverse side effect occur, and thus enhance compliance and reduce psychological distress.

TABLE III
PSYCHOLOGICAL FACTORS IMPORTANT IN THE ETIOLOGY OF ERECTILE DYSFUNCTION*

Developmental Factors
- unopposed maternal or paternal dominance (in two-parent home)
- one-parent family imbalance (one parent permanently absent due to death, divorce)
- overt mother-son sexual encounters
- conflicted parent-child relationship
- negative familial attitudes toward sexuality
- repressive influence of religious orthodoxy regarding sexual expression
- traumatic childhood sexual experience (*e.g.* rape, incest)
- homosexual influence and orientation based on early meaningful homosexual relationships or sexual experiences during the teenaged years
- traumatic first coital experience (*e.g.* dehumanizing experience with prostitute)

Affective
- performance anxiety
- spectatoring (male preoccupation with his sexual function to the extent that he is a spectator to, rather than a participant in, his sensual experience)
- guilt
- poor self-esteem
- fears of the consequences of sex (hurting the female, hurting self, pregnancy, venereal disease)
- fear of continued erectile difficulty following isolated episodes of erectile failure (frequently due to fatigue, alcohol/drug intoxication, acute illness, stress, transient anxiety)
- depression or mania
- fears due to intrusive thoughts and fantasies which are "forbidden" (*e.g.*, fantasies about another woman, unacceptable practices, or homosexual ideas)

Interpersonal Factors
- inadequate sexual cues from partner
- anger, hostility, or distrust toward partner
- decreased physical attraction to partner (*e.g.* obesity, pregnancy, or a heterosexual partner when the man is actually a homosexual)
- poor communications with partner
- sex role conflicts
- divergent sexual preferences (*e.g.* regarding frequency, types, time of sexual activity)
- anxious over-concern with degree of partner's pleasure

Cognitional Factors
- negative attitudes and misconceptions about sex
- ignorance of sexual physiology, psychology, and techniques
- iatrogenic influences (*e.g.* inadequate/inaccurate information or negative attitudes from a consulted professional

* Adapted from reference 1.

In addition to consideration of medical disorders and medication effects, there must be an assessment of the patient's psychological functioning and personality characteristics. For any given individual, the particular constellation of psychosocial, cultural, environmental, and experiential factors involved may vary considerably and often involves an interaction of multiple factors. Kolodny, Masters, and Johnson[7] classified these influences into four areas: developmental, affective, interpersonal, and cognitional. *Table III* summarizes the contributions of several authors[7-10] in regard to the important elements in each of these categories.

Evaluation Procedures

The comprehensive diagnostic evaluation currently being performed in the UKSM-W Sexual Medicine Clinic in the Department of Psychiatry includes the traditional detailed history, mental status examination, complete physical examination, psychological testing (Derogatis Sexual Functioning Inventory), baseline laboratory studies, and selected newly developed specialized studies: nocturnal penile tumescence (NPT) monitoring, Doppler ultrasonographic evaluation of penile blood flow dynamics, and when indicated, internal pudendal arteriography. *Table IV* summarizes the major relevant aspects of each of these components of the diagnostic evaluation.

One of the most promising diagnostic tools in recent years has been nocturnal penile tumescence monitoring. Sleep erections, described as early as 1940, are now known to be a normal phenomenon accompanying the "autonomic storm" of generalized increased body activity associated with rapid eye movement sleep and dreaming. The diagnostic usefulness of NPT monitoring is based on the assumption that during sleep, the psychological fac-

TABLE IV
UKSM-W SEXUAL MEDICINE CLINIC APPROACH TO THE EVALUATION OF ERECTILE DYSFUNCTION

History
- Elaboration of "chief complaint"; onset, situational context, presence or absence of nocturnal and morning erections, etc.
- Sexual history: early experiences/emotional reactions, attitudes toward sexuality, level of knowledge, frequency and types of sexual practices, acceptance of cultural myths, development of current sexual relationships, masturbatory practices and fantasies, homosexual experiences.
- Developmental and family history: death/divorce of parents, family attitudes toward sexuality, religious influence, relationship with parents/siblings; enuresis, etc.
- Marital history: development and current stability of the relationship, any changes in feelings toward partner, partner's reaction to the dysfunction, any extramarital affairs.
- Presence of current stressors: financial, occupational, etc.
- Medical history: presence of any illnesses, injuries, or surgery that may affect erectile function.
- Medications: current and previous.
- Habits: alcohol, illicit ("recreational") drugs, tobacco.

Review of Systems. General review, with particular attention to:
- Cardiovascular: exertional dyspnea, palpitations, claudication, cold extremities
- Neurologic: headaches, syncope, seizures, loss of consciousness, trauma, vision/smell/taste changes, paresthesias, loss of coordination, decreased strength, etc.
- Endocrine: intolerance to heat/cold, tremors, dry skin/hair, change in libido, polyphagia, polydipsia, polyuria, gynecomastia.
- Genitourinary: nocturia, polyuria, dysuria, hematuria, pyuria, urinary frequency or hesitancy, incontinence, history of venereal disease and any treatment, penile lesions or discharges, etc.

Mental Status
- Evidence of mood changes: depression, elation, lability; abnormal thought processes or content; perceptual distortions
- "Vegetative changes": disturbance in energy level, libido, appetite, sleep pattern, weight change
- Assessment of overall level of psychological and cognitive functioning

Psychological Testing
- Derogatis Sexual Functioning Inventory (DSFI)
- Beck Depression Inventory

Physical Examination, with special attention to:
- General: Vital signs, obesity, cachexia
- Cardiovascular; bruits (especially femoral), peripheral pulses, evidence of venous stasis, arterial insufficiency (especially lower extremities), palpation of epigastrium (to check for pulsatile mass)
- Neurologic: Gait, coordination, DTR's reflexes (especially lower extremities), pathological reflexes, sensation, motor strength; assess integrity of sacral reflex arc (S2-S4) with perianal sensation, anal sphincter tone, bulbocavernosus reflex
- Genitalia: size, consistency of testes, penis size/malformations/lesions

Baseline Laboratory Studies
- Complete blood count, Multi-Chem, urinalysis, fasting blood sugar, VDRL, thyroid profile
- Serum testosterone (drawn in AM)
- Serum prolactin, follicle-stimulating hormone, luteinizing hormone

Specialized Studies (when indicated)
- NPT (nocturnal penile tumescence) monitoring
- Penile blood pressure and pulse volume recordings (Doppler ultrasonography) pelvic and internal pudendal arteriography

tors impeding erectile function during wakefulness are no longer operative, allowing a demonstration of the integrity of one's physiologic capacity. Physiological deficits, however, would persist during sleep, and therefore inhibit the number and duration of erectile episodes.[11-13] Subsequent research in several centers has tended to confirm the validity of this assumption, with two exceptions: first, in certain psychiatric disorders (*e.g.*, endogenous depression) in which REM sleep patterns are also disrupted;[11, 14] and second, in a few men with organically proven erectile failure who occasionally have an episode of full erection during sleep, such as in patients with lower body spasms due to spinal cord injury, patients with a vascular "steal" syndrome, and in a previously unrecognized syndrome of impaired penile tumescence in the presence of sleep apnea, hypoventilation with decreased oxygen saturation, myoclonic jerks, and bradycardia.[14]

The protocol of the Wesley Medical Center Neurodiagnostic Laboratory for NPT monitoring requires two successive nights of polysomnographic monitoring. Parameters measured include EEG and EOG (to document presence and duration of REM sleep), heart rate, respiratory rate, descriptions of sleep pattern and body movements, changes in penile circumference at the tip (just proximal to the glans) and base using two strain gauges. Since increases in circumference may not necessarily be accompanied by sufficient arterial inflow to provide adequate rigidity for intromission,[15] an assessment of penile rigidity is also made during at least one of the erectile episodes, generally on the second night of monitoring. Rigidity is measured using a specifically designed tonometer which quantifies the amount of force required to "buckle" the erect penis.[15] The patient is briefly awakened when the rigidity challenge is performed, and asked to evaluate the quality of this erection, and to estimate its sufficiency for penetration. The technician also records his estimate of the degree and rigidity of the erection. A photograph of the erect penis is taken, and later reviewed with the patient. This photograph provides visual evidence of normal erectile capacity to the patient with psychogenic dysfunction; it aids in the interpretation of numerical data obtained; and it reveals or confirms the presence of any anatomical deformity interfering with normal erection and penetration. The data obtained from the NPT assessment are then summarized and reviewed with the patient during a followup visit to our clinic.

NPT measurements do not differentiate the many types of medical disorders which may impair erectile function.[13] Following an abnormal NPT study, additional specialized diagnostic studies are necessary to determine the exact mechanism(s) contributing to the dysfunction. These may include abdominal computerized axial tomography or myelography if there is evidence of neurological impairment, or if a vascular etiology is suspected on the basis of clinical and NPT findings, Doppler ultrasonography of the penile vessels or internal pudendal arteriography may be indicated.[2, 16-18] Velcek[18] has described the procedure for measurement of penile flow index using Doppler ultrasonography. Briefly, this procedure involves using a digital cuff and Doppler ultrasound probe to record systolic blood pressure and pulse volume in each of the penile arteries. The penile systolic blood pressure is then compared to the brachial systolic blood pressure to arrive at a "penile index." A penile index <0.9, or a penile blood pressure >30 mmHg *below* brachial systolic blood pressure is suggestive of vascular insufficiency as the etiology of impaired tumescence.[18] Internal pudendal arteriography may then be useful to delineate the extent of vascular obstruction and the degree of collateral circulation.[2, 9, 17, 18] Velcek[18] and Gray[2] have reported a high correlation between abnormal Doppler studies of penile blood flow, the presence of pelvic arterial disease on arteriography, and the clinical manifestations of erectile failure, but additional research is needed to develop appropriate criteria for selection of patients for arteriography given the morbidity of the procedure.

Gray[2] reports that of the two screening procedures used to select patients for angiography — NPT and Doppler studies — the ultrasound evaluation was the least reliable. He hypothesizes that if one good pulse is present, transmitted pulses may be auscultated in all four quadrants, leading to a high false negative rate for the Doppler procedure. In the series, all abnormal Doppler studies were in patients with abnormal angiograms, indicating that an abnormal Doppler study has a greater clinical value than a normal one in selecting patients for arteriography. These authors concluded that NPT monitoring, which is easy, accurate and noninvasive, provided a better screening procedure in selecting patients for angiography.

Treatment Approaches

A number of successful new modalities for treatment of erectile failure have been developed within the past decade. These have been extensively reviewed elsewhere,[1, 19, 20] and will only be mentioned here. Since the early 1970s, with the development of a variety of semirigid and inflatable penile prostheses, surgical management has become an

acceptable and established mode of therapy in properly selected patients. Numerous studies have shown that both types of prostheses provide sufficient rigidity for intercourse, are well tolerated, have comparable complication rates, and are satisfactory in increasing sexual enjoyment.[1]

Additional surgical treatment modalities include revascularization of the penis by implantation of the inferior epigastric artery into the corpus cavernosum for patients with small vessel disease,[17] and bypass grafts from the femoral artery to the corpus cavernosum.[2, 17] For patients with occlusive-vascular disease of the aortoiliac system, conventional endarterectomy and aortoiliac or aortofemoral grafting may restore erectile function.[16, 17] There may also be a role for percutaneous transluminal angioplasty in selected patients with large vessel disease. Gray[2] illustrates one patient with stenosis of one internal iliac origin in whom erectile function returned following transluminal angioplasty with resulting increase in luminal diameter and blood flow.

Medical strategies are aimed at correction of the underlying disorder (*e.g.* diabetes mellitus) or manipulation of medication thought to be affecting sexual function. At times, administration of exogenous testosterone has been advocated in the treatment of erectile dysfunction. Its efficacy, however, has not been established except possibly in patients on renal dialysis and in the few patients in whom serum testosterone is low without demonstrable cause (*e.g.* hyperprolactinemia).[10] Empiric administration of testosterone is not indicated, and may, in fact, result in increased desire for sexual activity without accompanying improvement in erectile function.[1, 10]

Psychotherapeutic treatment modalities are indicated when the erectile dysfunction is caused predominantly by psychosocial factors, and in organically determined dysfunction associated with a significant degree of psychological distress. There are many diverse psychological treatment models available: the "Masters and Johnson" behaviorally-oriented sex therapy for couples; Kaplan's psychodynamically oriented sex therapy for couples; psychoanalysis and psychoanalytically oriented individual therapies; brief individual psychotherapy; behavior therapy; and biofeedback training.[1] The selection of a particular approach will depend upon the relevant clinical details of each individual case, considering such factors as underlying psychodynamics and character traits, presence of other sexual dysfunction, intensity of the anxieties and fears involved, severity of associated relationship difficulties, lifestyle, motivation for change, and duration of the symptom.

Prior to the initiation of psychotherapeutic approaches, it is extremely important to evaluate the patient fully for the possibility of unappreciated organic disease.

In summary, there have been in the past decade many technological advances in the development of diagnostic procedures and treatment methods for erectile dysfunction. It is important for physicians to inquire about this aspect of their patients' lives, to be knowledgeable about available diagnostic and treatment methods, and to make appropriate referrals for comprehensive evaluation when warranted.

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Appointment-Keeping Compliance

Unexpected Benefits From an Unorthodox Program

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NONCOMPLIANCE with appointment-keeping is an ongoing problem in the treatment of hypertension. Besides the obvious loss of benefit to the patient, the cost and disruption caused by missed appointments is significant to a clinical practice. Studies estimate the dropout rate in clinic attendance for hypertension to be more than 50 per cent within the first year.[1]

However, the appointment that individuals most frequently fail to make, or break, is the first one following referral for care, either from within the medical care system (between physicians) or from outside the system, as from a screening clinic. Loss of the individual at this initial stage is disheartening: "the fight against his disease is given up without a single battle."[2]

In reviewing the compliance research literature, Sackett and Snow note that the results suggest ". . . that when appointments are initiated by health professionals, compliance with appointment-keeping is only about 50 per cent; the exceptions occur when children are involved. On the other hand, when the appointment is initiated by the patient, compliance rises to roughly 75 per cent." This same trend was also noted in an earlier review by Haynes and Sackett.[3]

The intent here is to briefly record the surprising effectiveness of nonhealth professionals in providing blood pressure screening and referral and to report the program design to Kansas physicians as a possible one to establish in their own community. Unexpected benefits of a new program, "REACH" (Respond Early and Control Hypertension), utilizing Wichita fire stations as blood pressure screening sites, were (1) maintenance of appointment-keeping compliance demonstrated by patients referred to community physicians by the firefighters as compared to referral from nurses in a standard screening program; and (2) increased treatment alteration by physicians for referred clients.

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Program Background

In 1973, the University of Kansas School of Medicine-Wichita (UKSM-W), in partnership with the Sedgwick County Medical Society, responded to the national challenge to detect and counsel individuals found to have high blood pressure by establishing a hypertension screening and education program for Wichita-Sedgwick County citizens. Through the administration of the Division of Health Care Outreach, a program was developed which provided hypertension screening services to those individuals who might not seek assistance from private sources. In keeping with this goal, emphasis has been placed on assisting targeted minority populations and older citizens. While target populations varied, the number of individuals screened annually has ranged from 1,200 in 1973 to a high of 20,359 for the 1981 program year. The total number of individuals screened during the past nine years of operation is more than 124,000.

Funding limitations for hypertension screening services in Sedgwick County resulted in development of a program utilizing neighborhood fire stations and firefighters. Abnormal readings are rechecked and followed up by UKSM-W nurses. Followup data indicate a comparable success rate for patients seeking medical care after referral from these nonhealth professionals as from health providers.

Until 1983, these screenings have been conducted by RNs and LPNs in a wide variety of community sites in Sedgwick County, utilizing a 32-foot Mobile Screening Unit or an "on-site" screening station. Past screening protocol provided that after an elevated reading was detected at an initial screening and confirmed by a recheck by the UKSM-W staff at a later date, the individual was referred to his/her private physician with subsequent telephone followup by the Outreach nursing staff. The standardized protocol for screening and followup, along with data

from the screening and followup component for the first eight years (1973-1981) have previously been reported.[4]

However, due to shifts within federal allocations (from 317a Public Health Service funds to state directed block grant funds), financial support for the hypertension project greatly diminished, necessitating a reassessment of screening methodology and allocation of paid staff time. Alternative strategies for establishing a reliable core of routinely available screening "volunteers" were developed. Subsequent discussions with the Wichita Fire Department and the Sedgwick County Division, Kansas Affiliate of the American Heart Association were initiated. This new program, REACH, was designed to permit UKSM-W to concentrate its resources on the follow-up of individuals with elevated blood pressure readings, as well as ensure a continuation of screening services to those in need. After approval of the program concept by the three agencies, protocols were designed, training provided to the firefighters, and screening activities intitated on January 4, 1983.

Methodology

The REACH program provides free hypertension screening services performed by on-duty firefighters at each of the 17 city fire stations, every Saturday from 10 A.M. to 2 P.M. Individuals, upon arrival at the fire stations, are directed to a specific quiet, non-distracting location. They are seated for at least five minutes and asked to complete a screening data form. Next, blood pressure is measured by a firefighter according to protocol established by the American Heart Association, as approved by UKSM-W. Results are then recorded on the screening form as well as on a card given to the individual. If an elevated reading is found, the individual is advised to return to the fire station for a recheck on the following Saturday, or to attend a recheck clinic held at UKSM-W every Thursday. If an elevated reading persists after a recheck, the individual is referred to his/her private physician. *Table I* displays the screening and referral criteria and timing of followup contacts.

TABLE I
PROJECT REACH: RESPOND EARLY AND CONTROL HYPERTENSION

Screening, Referral and Followup Guidelines

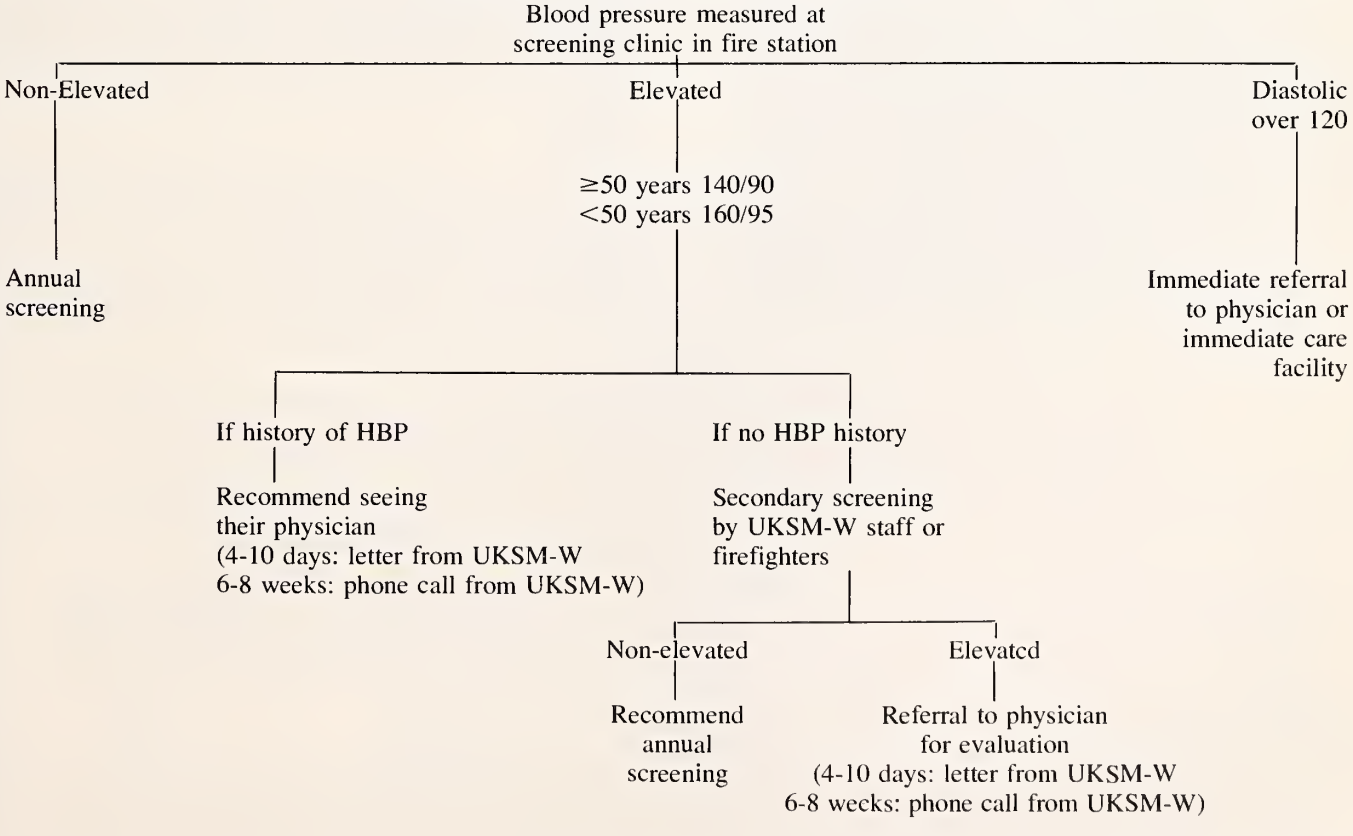


TABLE II
PROJECT REACH:
CHARACTERISTICS OF INDIVIDUALS SCREENED
(INITIAL VISIT)
January-July, 1983

Characteristics	*Percentage*	*Total Respondents Per Category*
I. Blood Pressure Status		
a. Elevated	29.4	
b. Unelevated	70.6	2,663
II. Age		
a. 15 years & below	4.06	
b. 15-19	.74	
c. 20-34	11.28	
d. 35-54	22.49	
e. 55-64	26.92	
f. 65 and above	38.16	2,571
III. Sex		
a. Female	52.9	
b. Male	47.1	2,642
IV. Ethnic Identification		
a. Caucasian	83	
b. Black	14	
c. Hispanic	2.2	
d. American Indian	.4	
e. Indochinese	.2	
f. Other	.2	2,567

Each of the 385 firefighters performing the blood pressure measurements participated in a two-hour training session involving both lecture and practice on approved procedures and protocols of blood pressure measurement. Their performance was evaluated by using teaching stethoscopes and completion of a written test. The training sessions and evaluation were provided by the medical training officer of the Wichita Fire Department. Wall mounted mercury sphygmomanometers with a variety of cuff sizes were purchased by the Wichita Fire Department. Equipment is checked regularly for proper calibration and function.

The completed screening forms are routed weekly to the UKSM-W nursing staff who then proceed with the followup procedures to encourage appointment-seeking and appointment-keeping behavior for individuals with elevated readings. Two contacts are made, one at four to ten days after the initial elevated reading (primary followup) and another at six to eight weeks after referral to the physician (secondary followup). The primary followup through letter encourages clinic attendance while the secondary followup provides encouragement to those who have not attended, or to reinforce physician directions for those who have attended. The timeline for this screening followup appears in *Table I*. Telephone contact has proven to be the most effective for the "secondary" followup. However, if contact is not made after three attempts by phone, a letter is sent requesting a response on an enclosed self-addressed postcard.[5]

The Sedgwick County Division of the Kansas Affiliate of the American Heart Association provides funding for printing of program forms and publicity materials, staff assistance in program coordination, patient education materials on hypertension in a literature rack at each fire station, and volunteers to tabulate the screening data on a weekly basis.

Results

A total of 3,430 client visits were made during the first six months of the project (January-July 1983) with 2,663 individuals seen on an initial visit for blood pressure screening and 767 subsequently re-

TABLE III
REACH PARTICIPANTS:
HISTORY AND TREATMENT STATUS OF HYPERTENSION UPON INITIAL VISIT
January-July, 1983

Blood Pressure Reading Upon Initial Visit	*Reported History and Treatment Status*		
	History of HBP Currently on HBP Treatment	*History of HBP No HBP Treatment*	*No History of HBP No HBP Treatment*
Elevated (29.4%, n = 784)	17.5% (n = 465)	3.7% (n = 98)	8.3% (n = 221)
Non-Elevated (70.6%, n = 1,879)	29.2% (n = 778)	7.0% (n = 185)	34.3% (n = 916)

Total n = 2,663

TABLE IV
PARTICIPANTS IN PAST UKSM-W COMMUNITY SCREENINGS
HISTORY AND TREATMENT STATUS OF HYPERTENSION UPON INITIAL VISIT
July 1979-June, 1980

Blood Pressure Reading Upon Initial Visit	*Reported History and Treatment Status*		
	History of HBP Currently on HBP Treatment	*History of HBP No HBP Treatment*	*No History of HBP No HBP Treatment*
Elevated (12.6%, n = 1,057)	3.8% (n = 322)	3.2% (n = 272)	5.5% (n = 463)
Non-Elevated (87.4%, n = 7,344)	15.8% (n = 1,329)	10.0% (n = 844)	61.5% (n = 5,171)

Total n = 8,401

turning to the fire stations for rechecks or monitoring. This enthusiastic response indicates an acceptance of REACH by the community, as well as by the physicians who are referring patients back to the fire stations for regular monitoring.

Of those attending for their initial visit, 29.4 per cent (n = 784) were found to have elevated readings. This is higher than that found in previous community wide Sedgwick County screenings.[4] Additional data describing age, sex, and ethnic identification for this group are summarized in *Table II*.

The reported treatment status and history of hypertension, crossed by the variables of elevated and unelevated readings, are displayed in *Table III*.[4] The same variables reported for the 1980 program year, during which time UKSM-W nurses conducted screenings in a variety of community locations, are displayed in *Table IV*. Significant differences between the two approaches include: (1) the percentage of participants with an elevated reading reporting a history of hypertension and currently under treatment with 3.8 per cent in 1980 as compared to 17.5 per cent in 1983 ($p \leq .05$); (2) the percentage of participants with an unelevated reading who report history of hypertension who are currently under treatment with 15.8 per cent in 1980 as compared to 29.2 per cent in 1983 ($p \leq .05$); and (3) the percentage of participants with an unelevated reading who report no history of hypertension with 61.5 per cent in 1980 as compared to 34.3 per cent in 1983 ($p \leq .05$).

The data comparing the REACH project with the

TABLE V
COMPARISON OF OUTCOMES REPORTED UPON SECONDARY FOLLOWUP
FOLLOWING REFERRAL TO PHYSICIAN

Outcome Reported Upon Secondary Followup	*1982**	*1983***
I. Physician seen: attended clinic appointment		
a. following same medication regime	17 %	5.6%
b. placed on alternative treatment plan		
c. treatment was revised	28 %	44.9%
d. did not follow with treatment plan	3 %	1.2%
e. not started on medication	19 %	16.2%
f. directly admitted to hospital	.3%	1.2%
g. started following previous prescribed		
II. Has not seen physician	19 %	20.6%
III. All other individuals (unable to locate, etc.)	10.7%	1.2%
Total	(100%) n = 581	(100%) n = 247

* Data reflecting hypertension screening done in a variety of non-repetitive community sites by nurses

** Data reflecting hypertension screening done entirely by firefighters in Wichita fire stations on a routine, weekly basis through project REACH

previous screening project differs significantly when examining the client and physician behavior after a referral from a firefighter due to an elevated reading. *Table V* displays the outcome of those with elevated readings from the 1982 UKSM-W screenings, as well as data collected from the REACH program (1983). As previously mentioned, after confirmation of the elevated reading during a recheck, clients were referred to physicians for followup. A letter encouraging them to seek an appointment was sent during the next four to ten days. After a six to eight week time lapse, the UKSM-W nursing staff contacts the client to assess the referral outcome or provide further client encouragement. In 1982, 70.3 per cent of individuals with an elevated reading made contact with their physicians, as compared to 78.2 per cent making contact with their physicians after referral from the REACH program in 1983. A significant point of variance between data from the two programs reflected physician behavior after referral: 44.9 per cent modified medications after a REACH referral, as compared to 28 per cent modifying medication after a referral from the 1982 program.

Discussion of Results

The enthusiastic response from both the public and professional communities, as well as the high level of public participation in the project should be mentioned. With a relatively small direct operating cost, a large number of citizens responded to the availability of the REACH program. It was particularly interesting to note the dedication of some of the individuals seeking blood pressure screening at the fire stations. On two Saturdays during February 1983, Wichita experienced a sudden snowfall of four to six inches. In spite of many businesses being closed and the municipal airport non-operational, 58 and 44 individuals (respectively) made it to their fire stations on those days to have their blood pressures checked. This overall participation was significant given the ''cognitive jump'' required by the participants in identifying the fire stations as someplace to ''visit'' for health services.

The REACH participants are different, however, from those seen in previous screening years, as reflected in *Tables III* and *IV*. The proportion of those with a history of elevated blood pressure, and who are on treatment, was 46.7 per cent for the REACH program, as compared to 19.6 per cent for those screened in the 1980 program year. This would indicate that the individuals who seek REACH services are more likely to do so because of a known concern about hypertension, and that the REACH data does not reflect the prevalence of hypertension within the Wichita population. This is predictable, given that visiting a fire station is an action involving intentional behavior, whereas participation in past screening involved more passive behavior with convenient measurements provided at community locations where a large number of people were already congregated (*i.e.*, festivals, grocery stores, worksites). Although the REACH population was more likely to have a history of hypertension, it is significant that 221 (8.3%) were detected as ''new'' hypertensives (no history, no treatment).

As the populations and screening methodology are different, it is not possible to draw comparative conclusions regarding appointment-keeping compliance after a referral from a firefighter versus referral from a nurse. However, it is remarkable that 78.2 per cent of those referred by a firefighter did see a physician within two months. Furthermore, it is significant to note that these were valid referrals given the high number (44.6%) of participants reporting a medication change prescribed by their physician during the physician office appointment following the referral.

Conclusion

The REACH Program is successful as evidenced through: evaluation of community wide participation; patient behavior in ''appointment-keeping'' after a referral by a firefighter to a physician; validation of need to be seen (physicians modifying treatment for those referred); and in credibility (number of physicians referring individuals back to the program for monitoring).

Furthermore, the initiative is a cost-effective one in maximizing the use of limited resources available, allowing UKSM-W nursing staff to concentrate on followup instead of actual screening. Firefighters, as indicated by the data, are effective in providing referrals, which benefits both clients and physicians as well as providing an excellent public relations program for the fire department. A concern develops, however, in examining the impact of the program on the minority and high-risk populations in Wichita. Although the percentage of minority participants of the total population screened is roughly equivalent to the percentage of minority individuals reported in 1980 census data, additional strategies for involvement need to be developed to further provide services to this high risk group.

Demonstrated efficiency would dictate that this program should be implemented within any community possessing adequate firefighter assistance

(Continued on page 499)



Cameo

THE SURGEON AND I, THE FAMILY DOCTOR

by Louis Forster, M.D.*, Salina

Time for him had collapsed
long before we bored into his chest
freeing
the two ribs on the right, sternum to backbone . . .
these we discard . . . and separating
the remaining ones

seeking in the air-fluid filled sponge
of lung the xrayed spot.

Soon the pathologist . . . amazing how
we see his face as a white
surgery room wall and mouth
as a metal intercom box . . .
says bad, repeat bad
as we knew he would.

Told the family we found it
(he the surgeon seeming the father
type, me the brother-sister-neighbor
next-door role)

knowing they already knew
sloshing coffee
in styrofoam cups
filled by a smiling
waiting room hostess.

I know I found myself saying,
saying,

before leaving to walk to
my office full of
waiting patients,
alone.

* Clinical Instructor, Department of Family and Community Medicine, The University of Kansas School of Medicine-Wichita, at Smoky Hill Family Practice Residency Program, Salina.



Digoxin-Quinidine Interaction

Clinical Considerations of Two Potential Mechanisms

PHILIP D. DYER, M.D., ***Fort Sam Houston, Texas***

CONCURRENT USE of digoxin and quinidine was questioned by Gold[1] in an article published in 1932; he was alarmed by the variability in effects of adding quinidine to digitalized animals. Recent concern has developed secondary to several reports in 1978 detailing a rise in serum digoxin after initiating quinidine therapy. This interaction has been manifested by increased serum digoxin levels determined by radioimmunoassay and by symptoms and signs of digitalis intoxication. The development of a radioimmunoassay for digoxin in 1969, by Smith *et al.*,[2] was an important contribution for the detection of this interaction. Using the assay on digitalized patients treated with quinidine, an average two-fold increase of digoxin has been reported secondary to the institution of quinidine therapy. The mechanism of interaction between digoxin and quinidine is currently being debated. Some investigators feel decreased renal clearance causes the digoxin elevation, while others propose displacement of digoxin at its binding sites by quinidine. Other antiarrhythmics have been evaluated to check possible interactions with digoxin. Digitoxin has recently been investigated and found to be elevated with simultaneous quinidine treatment. The above considerations have surfaced due to discovery of the digoxin-quinidine interaction; a review of current literature expands on the above points. Also, application of these findings to clinical practice will be discussed.

Before considering the rise of digoxin, it is important to consider the therapeutic range of digoxin and its low therapeutic-toxic ratio. The therapeutic range listed by Goodman and Gilman[3] is 0.5-2.0 ng/ml, and toxic effects can occur at levels above 2.0 ng/ml. With this consideration, the therapeutic-toxic ratio is approximately two-fold with the average therapeutic dose being around 1.1.[2] A two-fold value is significant because it is the average increase of digoxin during quinidine therapy. One of the first studies to illustrate this was done by Ejvinsson;[4] he studied 12 patients on maintenance digoxin who were loaded with quinidine and maintained on the equivalent of 1.2g of quinidine sulfate. The average digoxin level prior to quinidine treatment was .85 ng/ml and aver-

Concurrent use of digoxin and quinidine results in approximately a two-fold increase in the serum digoxin level, moving it into the toxic range. It is therefore suggested that digoxin dosage be reduced by one-half prior to administration of quinidine. Pending further research on the two possible mechanisms of interaction, ramifications of both should be considered in treatment of patients.

Editor's Note: Papers published in the Student Thesis series have been written by fourth year medical students at the University of Kansas School of Medicine. Those selected for publication have been recommended by the UKSM faculty.

age value during treatment was 1.6 ng/ml. One patient developed symptoms of digitalis intoxication when the plasma value rose above the therapeutic range. This study was one of the first, and its findings have been duplicated by other investigators.

Another contribution, published in 1978 by Leahey *et al.*,[5] showed significant rise of digoxin concentration in 25 of 27 patients being studied. All 27 patients had normal renal function and were started on quinidine therapy for atrial or ventricular arrhythmias. The mean serum digoxin concentration rose from 1.4 ng/ml to 3.2 ng/ml, slightly greater than a two-fold increase in the digoxin level. The rise to 3.2 ng/ml placed patients in the toxic range as reflected by symptoms of toxicity,[4] which will be studied later. Doering[6] also reported a greater than two-fold rise in digoxin levels in a study of 38 patients with normal renal function. Even though the digoxin level rose in 79 patients and five volunteers, only the 38 with normal renal function were considered. Their level increased from .98 ng/ml to 2.47 ng/ml, a 2.5-fold increase with a therapeutic dose of 1000mg of quinidine bisulfate. The final paper by Hager *et al.*[7] reported increased levels of digoxin in all six volunteers. His subjects received intravenous digoxin after being started on 800mg of oral quinidine sulfate daily and compared this to intravenous digoxin alone. A plot of digoxin concentration *vs* time for a representative subject showed a digoxin level of 1.0 ng/ml without quinidine and a level of 1.8 ng/ml post quinidine at ten hours after digoxin injection. Again a figure roughly two-fold in value estimates the expected rise in serum digoxin level. Considering this value, symptoms of digoxin toxicity would be expected and have been reported in several studies.

Reports of digoxin toxicity focus on the symptoms of the gastrointestinal system and signs of cardiac toxicity. Anorexia, nausea, and vomiting are among the earliest symptoms of digoxin toxicity and are secondary to irritation of the chemoreceptor trigger zone of the medulla.[3]

Cardiac toxicity is associated with disturbance of rhythm and A-V conduction. Disturbances of ventricular rhythm are premature depolarizations that may appear as bigeminy; ventricular tachycardia and ventricular fibrillation can also occur. Atrial rhythm disturbances include tachycardia and premature depolarizations; sinoatrial and atrioventricular block can occur along with A-V junctional tachycardia.[3] Leahey,[5] in his paper, reports 16 of 27 patients (59%) having symptoms of anorexia, nausea, or vomiting when started on quinidine. More than one-half (59%) showed evidence of toxicity as compared to well known estimates of 20 per cent in hospitalized patients on digitalis. Ten patients of the 16 continued quinidine therapy, and reduction of the digoxin dose resulted in disappearance of these symptoms in all ten patients.

Concerning cardiac arrhythmias, he reported six cases of advanced ventricular arrhythmias. Three cases of ventricular tachycardia developed plus ventricular fibrillation in one and asystole in another. The above arrhythmias developed secondary to the initiation of quinidine therapy in digitalized patients. Leahey,[8] in an earlier publication, discussed three cases of digitalis intoxication secondary to starting quinidine therapy. The first patient's level increased from 2.2 ng/ml to 4.1 ng/ml; the patient developed nausea, vomiting, and weakness. The patient also developed frequent ventricular premature depolarizations, and the ventricular response rate slowed from 90 to 45 beats/minute. The digoxin level rose from 1.9 ng/ml to 3.8 ng/ml in the third patient started on quinidine. He developed A-V junctional tachycardia at 142 beats/minute, which resolved 36 hours after discontinuance of digoxin. These two cases show two classic examples of digoxin induced arrhythmias, which resolved with decreased digoxin dose. These reports by Leahey show both gastrointestinal and cardiac manifestations of toxicity, which will be shown to be significant when discussing the displacement of digoxin from its binding sites.

When reviewing the literature on quinidine induced elevations of serum digoxin, the question arises concerning what dosage of quinidine is necessary and how rapidly elevation occurs. Limited investigations have been done on the topic; results show the digoxin level elevation is directly proportional to the quinidine dosage. Most research has been done at therapeutic doses of quinidine, in the range of 1000mg daily. Doering[6] found no significant elevation at daily doses less than 500mg. One patient accidently received 3000mg/day, and his level rose four-fold; the remaining patients receiving around 1000mg/day had the 2.5-fold increase. He also plotted digoxin concentrations *vs* time in days for five healthy volunteers on 1000mg of quinidine. The digoxin level rose on the second day and leveled off on the fifth; digoxin levels returned to normal five days after discontinuing quinidine.

Another report by Hirsh *et al.*[9] showed a 21 per cent increase in digoxin level after one day with only one 400mg dose of quinidine sulfate. Hirsh's group continued with quinidine at six-hour intervals for 24 hours, and their levels rose 59 per cent. A report by Risler *et al.*[10] showed a digoxin level increase from 0.75 to 1.2 on concomitant administration of 500mg

of quinidine sulfate/day in 11 healthy volunteers. With further increase in the dose to 1000mg, the digoxin level rose to 1.8. In one of his three case reports, Leahey[6] showed that the serum digoxin level rose within 36 hours of quinidine therapy and the interaction continued for three months until the quinidine was discontinued. The above reports lead to some generalizations about the interaction, the first being that digoxin level is dose dependent on quinidine. Another is that the interaction occurs within the first day or two and that continued quinidine administration results in continued interaction.

Before discussing the possible mechanism involved, note should be made of possible interactions of digoxin with other antiarrhythmics. Leahey[11] compared digoxin levels in 27 patients on therapeutic quinidine with 26 patients on procainamide. He found the digoxin levels in the quinidine treated patients rose from an average of 1.4 to 3.1, while patients treated with procainamide had no elevation with unchanged average values of 1.2. Doering[6] considered seven different antiarrhythmic drugs that ranged from lidocaine to verapamil; each drug was reviewed in at least three patients with a total of 39. He found no significant changes in serum digoxin values of any of the seven other antiarrhythmics.

In view of the difference in structure between quinidine and the other antiarrhythmics this result is not unexpected. Procainamide is similar to quinidine in its antiarrhythmic properties; Leahey reports no change in the digoxin level with procainamide. Therefore, quinidine is the only antiarrhythmic considered here.

One mechanism suggested as the cause for the rise in serum digoxin level is displacement of digoxin from tissue binding sites. Before exploring this mechanism, digoxin binding should be further clarified. The positive inotropic effect of digitalis is believed to result from the binding of digitalis enzyme to the Na^+, K^+-ATPase, a membrane bound enzyme.[3] The binding results in inhibition of the ATPase with inhibition of transport of the two cations Na^+, K^+. This causes an increase in intracellular Na^+; due to increased Na^+ there is an exchange of intracellular Na^+ for extracellular Ca^{++}, and presumably increased intracellular Ca^{++}. Calcium's importance in muscle contraction is well known, but how increased calcium leads to improved myocardial contraction is questioned, with several theories being debated.

Other consideration of digoxin specifically is its 25 per cent protein binding, allowing for greater tissue distribution.[3] Digoxin is widely distributed to skeletal muscle with its concentration here being much higher than plasma levels. Its concentration is highest in the myocardium, being about twice that of skeletal muscle. Digoxin binding involves concentrated binding to the ATPase in the myocardium with a large volume of distribution, especially to skeletal muscle — skeletal muscle being a depot for digoxin.

The questionable mechanism of displacement of digoxin by quinidine at digoxin binding sites was initiated by Straub *et al.*[12] who demonstrated reduction of the number of digitalis binding sites by quinidine. Straub preincubated beef myocardium with Na^+, K^+ ATPase and quinidine, and ATPase alone as a control. When adding tritiated (H3) ouabain to these preparations, he found a significant decrease in binding of the digitalis to the beef myocardium in the quinidine treated preparation. He concluded that quinidine alters digoxin binding which causes digoxin serum elevation.

Straub[13] again demonstrated decreased tissue binding utilizing dog skeletal muscle. He gave digoxin to five dogs and then added quinidine; six dogs received digoxin alone. The addition of quinidine caused an increase in serum digoxin levels as well as vomiting and cardiac irregularities. After the animals were killed, the muscle tissue of quinidine treated dogs showed a digoxin concentration of 17.65 ng/gm; in those without quinidine, the level was 25.1. Since the dogs were already on maintenance digoxin, this suggests displacement of digoxin by quinidine in the depot of skeletal muscle.

Doering[6] tried to duplicate this finding; he used sarcolemma fraction of lamb myocardium and preincubated the tissue with quinidine, then added tritiated (H3) ouabain. He found no alteration, in the presence of quinidine, of digitalis binding. Later Straub[14] wrote that this failure was secondary to lack of pretreatment with Na^+, K^+ ATPase, and this alone accounted for Doering's failure to duplicate his findings. This finding by Straub was the initiation of the argument in favor of the displacement theory; he provides two valid investigations in support of this idea.

Another point that gives favor to this argument is recent reports of decreased volume of digoxin distribution following quinidine therapy. The decreased volume of distribution would result from displacement of digoxin from sites in muscle and myocardium; fewer sites would give higher serum levels and smaller apparent volume of distribution. Hager[7] used six adults to develop 12 curves of digoxin concentration *vs* time, a curve of controls, and one curve with quinidine treatment. He calculated the volume of distribution from each curve and arrived at an average of 10.87 l/kg for digoxin, which de-

creased to an average of 7.35 l/kg with quinidine added.

A more recent publication by Hirsh[9] also gave support to displacement of digoxin. He found decreased digitalis effect on the heart and concluded there was displacement by quinidine. Hirsh used systolic time intervals to evaluate nine healthy humans on digoxin and then on digoxin and quinidine. He found the expected rise in serum digoxin levels, but the systolic time intervals were increased with quinidine. This shows a negative inotropic effect despite increasing serum digoxin levels. Because the negative inotropic effect was greater than expected by quinidine alone, Hirsh thought that quinidine must be displacing digoxin from tissue-binding sites in the myocardium.

This argument should be taken lightly since there are no cases reported of cardiac failure in digitalized patients started on quinidine. Yet, there are several different reports on cardiac digitalis intoxication in humans and animals when quinidine was instituted. These reports counter the displacement mechanism because cardiac toxicity reflects more binding of digoxin to cardiac tissue. One could suggest a different binding site for cardiac toxicity and allow decreased binding at the Na^+, K^+ ATPase. With no reports of cardiac failure with quinidine, this idea would be only speculative. Therefore, the displacement mechanism develops from Straub's research of decreased binding sites and other reports of decreased volumes of distribution.

Another mechanism has been proposed resulting in a controversy between the two; the second mechanism involves a decrease in renal clearance causing the increased serum digoxin. Goodman[3] lists digoxin as 25 per cent protein bound with a urinary excretion of 72 ± 9% — the drug being filtered at the glomerulus and secreted with some reabsorption. Quinidine excretion in the urine is only about 20 per cent; most is metabolized by the liver.

The first report of decreased renal clearance was published in 1978. Hooymans[15] and Merkus presented three patients on digoxin who had 24-hour urine collections during and after quinidine therapy. All three patients had significant decreases in digoxin clearance during quinidine therapy; creatinine clearance remained constant. Hooymans postulated that quinidine causes a decrease in renal clearance of digoxin and that explained the elevated serum digoxin levels. Risler[10] used 11 volunteers to demonstrate a decreased renal clearance from 186.2 ml/min/1.73m^2 to 125.4 on a quinidine dosage of 500 mg/day. Increasing the quinidine dose to 1000mg a day resulted in a clearance averaging 120.6, with creatinine clearance remaining constant. He concluded that elevation of digoxin is due to the fall in renal clearance, but also suggested that the kidney might be saturable.

Other investigators have found similar results concerning the change in renal clearance. Hager *et al.*[7] found renal clearance fell from 1.64 to 1.09 ml/min/kg with initiation of quinidine therapy to his six volunteers. Doering[6] used 13 patients with normal renal function to show a decline of renal clearance from 91.6 ml/min to 40.6. He used 24-hour urine collections and midpoint blood samples. The subjects received maintenance doses of quinidine at 1000mg/day; the creatinine clearances also remained constant. With creatinine clearances constant, the decreased renal clearance could be secondary to decreased tubular secretion or increased renal reabsorption and not to changes in glomerular filtration.

Another interesting point was made by Lee;[16] he says decreased renal clearance would cause persistent digoxin elevations on unchanged dosages. This sustained effect was shown by Leahey,[8] and it lasted more than three months. Lee predicts that if displacement were involved, the elevation would be transient, following a new steady state of tissue binding. Doering[17] agrees with this point and says that volume of distribution changes by displacement would cause only transient changes in digoxin levels. This mechanism of decreased renal clearance is supported by several independent investigations with similar results; digoxin's 75 per cent renal clearance is also an important consideration. With the above factors considered, decreased renal clearance becomes a more plausible mechanism.

With two quite different mechanisms proposed and little research completed on the subject, a debate has developed regarding the more plausible mechanism. The January 17, 1980, issue of the *New England Journal of Medicine* published three letters supporting the renal clearance mechanism and three for the displacement theory. Most authors did compromise and suggest that, while their mechanism was the contributing factor, the other mechanism could be partially responsible. The mainstay of the displacement argument is the finding by Straub of decreased binding sites and several reports of decreased volumes of distribution.

The in vitro experiments by Straub have not been confirmed. Also, it would be difficult to explain observations of cardiac toxicity secondary to quinidine therapy in view of the displacement proposal. The renal mechanism is proposed because of several

consistent reports of decreased renal clearance of digoxin. It is thought that the 20 per cent renal excretion of quinidine competes with digoxin secretion and causes the serum elevation. This would lead to the reports of both gastrointestinal and cardiac digitalis toxicity — reports commonly found in literature on the digoxin-quinidine interaction. Both mechanisms have valid support, but clinical observation of digitalis cardiac toxicity can only support the renal clearance proposal.

The existence of two entirely separate mechanisms does not bar the possibility of coexistence. Hager[7] calculated the elimination half-life of digoxin and found it did not change when quinidine was administered. He also found a decreased renal clearance and decreased volume of distribution. Bigger[18] pointed out in a recent editorial that total clearance (Cl) equals the rate constant of elimination (Ke) multiplied by the apparent volume of distribution (Vd). Since the half-life is constant, Ke is constant and both the clearance and volume of distribution must decrease. $Cl = Ke \cdot Vd$. This illustrates that both mechanisms could contribute to the rise in serum digoxin with a constant half-life. Bigger suggests that both mechanisms are probably involved, and until further research is completed, one must suspect consequences of either mechanism. He concludes that one must watch for signs either of too much or too little digoxin.

The interaction of increased digoxin levels with quinidine is well documented, but information of the mechanism responsible is limited. Renal clearance seems to be the dominating theory because of reports of digitalis cardiac toxicity. Gastrointestinal intoxication can occur via both mechanisms, and itself requires reduction of digitalis dose. Because quinidine causes roughly a two-fold increase of serum digoxin, a one-half reduction of digoxin is warranted. This reduction in dose will help alleviate toxic symptoms. Until the displacement mechanism is proved insignificant or invalid, one must watch for signs of digitalis insufficiency with a reduced dose.

If the interaction occurs with digoxin, would the same interaction occur if digitoxin were substituted for digoxin? Digitoxin differs from digoxin by a hydroxyl group which results in different pharmacological properties. Digitoxin is 90 per cent protein bound, major elimination is hepatic metabolization, volume of distribution is smaller, half-life is nearly seven days, and therapeutic level is nearly 20 times higher — 10-35 ng/ml.[3]

Storstein[19] published the earliest report of digitoxin; he found the level of digitoxin increased only from 19.0 to 19.9 ng/ml in seven subjects during 24 hours following introduction of quinidine. He concluded quinidine did not elevate digitoxin levels, but investigations with digoxin noted a significant rise only after two days of concurrent treatment. Due to the length of Storstein's observations, his results are inconclusive. Two recent publications show digitoxin interacting with quinidine producing serum elevations as with digoxin. Fenster[20] used five healthy adults and plotted a semilogarithmic plot of digitoxin concentration *vs* time. His graph continued for 420 hours, and showed a significant elevation in digitoxin in the presence of quinidine in all five subjects. The elevation became more pronounced as time increased.

Garty[21] showed identical results by plotting digitoxin concentrations for five healthy subjects; his plot covered 26 days, which was necessary because of the increased half-life of digitoxin with quinidine. Fenster and Garty have shown elevation of digitoxin similar to digoxin, but the mechanism of serum elevation needs further consideration.

The two digitoxin researchers found identical results regarding the pharmacokinetics of the digitoxin-quinidine interaction. Both found the half-life increased to well over 200 hours with the reported value around 160 hours.[20, 21] The volume of distribution remained the same, but the total body clearance decreased in both reports. Fenster showed the renal clearance decreased from 0.65 to 0.46 ml/hr/kg. These results show the mechanism with digitoxin centers with decreased elimination; renal clearance is reduced and decreased liver clearance must also be considered.

Digitoxin is mainly cleared by the liver and its dose is unchanged during renal failure, which signifies the importance of hepatic clearance in total body clearance. The effect of quinidine on hepatic clearance must be evaluated to further clarify the decreased body clearance responsible for the rise in digitoxin. The constant volume of distribution indicates no displacement of digitoxin from its binding site, unlike the interaction involving digoxin. Fenster[20] comments on the higher lipophilic nature of digitoxin and its resulting higher binding affinity. This difference may account for the lack of displacement of digitoxin by quinidine. With decreased clearance, the half-life increases, the serum digitoxin level rises, and the potential for toxicity becomes a major consideration. Therefore, digitoxin does not constitute a therapeutic alternative for digoxin when quinidine treatment is warranted. The digitoxin serum level will increase with quinidine, due to decreased whole body clearance. Displacement does not play a role in this interaction.

With digitoxin considered along with digoxin, the scope of the digoxin-quinidine interaction is completed by reviewing the recent literature. Concurrent use of digoxin and quinidine causes concern due to a roughly two-fold increase in serum digoxin levels. The interaction occurs on therapeutic quinidine doses and appears to be directly related to the quinidine dose. Other antiarrhythmics do not cause a similar interaction. The rise in serum levels has clinically produced a gastrointestinal and cardiac digitalis toxicity — toxicity that can be resolved by lowering the digoxin dose. The mechanism of interaction is debated between decreased renal clearance of digoxin and displacement of digoxin at its binding site.

More recent suggestions involve both mechanisms together, the combination of the two being responsible for the elevations. Even though the renal clearance mechanism is supported by clinical observation, both should be considered clinically when considering possible complications of the interaction. Digitoxin was also reviewed to consider a possible alternative, but similar elevation of digitoxin eliminates digitoxin as a substitute. The digitoxin elevation is secondary to decreased whole body clearance and with this limited information, little can be concluded about the digitoxin-quinidine interaction.

The decision that can be made is the need to decrease the digoxin dose when initiating quinidine therapy. The suggestion of one-half the regular maintenance dose is sensible and should result in therapeutic digoxin levels. Determinations of serum digoxin levels should be done following this change along with periodic electrocardiograms. The reduction of the dose should control the development of digitalis intoxication, but until the mechanism responsible is proven, signs of digitalis insufficiency should also be considered.

Digitalis intoxication should always be considered in patients taking digoxin due to well documented reports of occurrence in hospitalized patients, but the above recommendation should prevent any increase in intoxication with quinidine therapy. Other factors that are important in digitalis therapy should also be included; low potassium and magnesium, high calcium levels, and the thyroid state can all complicate digitalis therapy. One final consideration: if quinidine therapy is discontinued, one must remember to increase the digoxin dose to the prequinidine maintenance dose. These clinical recommendations are made in light of the current information available on the digoxin-quinidine interaction. Greater knowledge of the interaction gained from continued research may result in further recommendations and suggestions.

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Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Sjögren's Syndrome — An Update

EARL ROONEY, M.D.* *and* HERBERT B. LINDSLEY, M.D.,† *Kansas City, Kansas*

THE CLINICAL manifestations of Sjögren's syndrome first were reported in 1888 by Hadden at a meeting of the Clinical Society of London. He described a 63-year-old widow with a dry mouth and a deficiency in lacrimal secretion. Numerous papers followed, which associated arthritis with various complaints of dry eyes and dry mouth. In 1933, Henrik Sjögren published a classic monograph that combined the clinical and pathological findings into a single entity and introduced the term keratoconjunctivitis sicca to describe the associated eye characteristics.

Sjögren's syndrome may be defined as a chronic inflammatory systemic autoimmune disorder, characterized by a triad of keratoconjunctivitis sicca, xerostomia, and connective tissue disease *(Table I)*. The diagnosis can be made from any two of these three major features. Sjögren's syndrome is classified as primary when only the first two criteria — keratoconjunctivitis sicca and xerostomia — are present. Secondary Sjögren's syndrome refers to the association of either keratoconjunctivitis sicca or xerostomia with a connective tissue disease, most commonly rheumatoid arthritis *(Table II)*.

Sjögren's syndrome, in both forms, occurs primarily in women. The disease has been reported in all age groups, including childhood, but the onset most frequently occurs in adults with a peak between 40 and 60 years.

The cause of Sjögren's is not known; however, immunologic abnormalities have been characterized extensively and are believed to contribute substantially to the pathogenesis of this disorder *(Table III)*.

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Ocular Manifestations

Keratoconjunctivitis sicca refers to the destruction of corneal and bulbar conjunctival epithelium as a result of decreased or absent subconjunctival gland secretion. Symptoms include burning of the eyes, foreign body sensation, dryness, itching, decreased vision, or photosensitivity. Objective findings may be determined with Schirmer's test, rose bengal or fluorescein staining, and slit lamp examination. Schirmer's test (a thin strip of paper placed in the lower conjunctival fornix) is considered abnormal with less than 10 mm wetting at five minutes. Test results vary considerably due to variations in temperature, humidity, and the age of the patient.

Fluorescein and rose bengal solutions stain dead or damaged cells and denuded areas of the cornea; dry spots and a rapid break-up in the tear film also may be seen with a slit lamp. Corneal epithelial filaments described as filamentary keratitis may also be seen. Complications of keratoconjunctivitis sicca include corneal abrasions, ulcerations, and infections.

Oral Manifestations

Xerostomia, or dry mouth, is a common symptom of Sjögren's syndrome *(Table I)*. Other symptoms include oral fissures and ulcerations, increased dental caries, difficulty with mastication, and loss of taste. The mucous membranes may appear dull and atrophic; lips may dry and crack. Parotid gland enlargement occurs in approximately one-third of patients. Since xerostomia commonly is seen in disorders other than Sjögren's syndrome, more specific tests have been developed to evaluate salivary gland involvement:

TABLE I
SIGNS AND SYMPTOMS OF SJÖGREN'S SYNDROME

Ocular
Dry, burning, itching eyes
Abnormal Schirmer's test
Abnormal slit lamp examination
Oral
Dry, atrophic oral mucosa
Oral fissures and ulcerations
Difficulty with mastication
Parotid gland enlargement
Systemic
Respiratory — nasal dryness, hoarseness, recurrent bronchitis
Skin — dryness, decreased sweating
Renal — renal tubular acidosis, nephrogenic diabetes insipidus
Musculoskeletal — arthralgia, myalgia, Raynaud's phenomenon
Neurologic — trigeminal neuropathy
GI — dysphagia, hepatomegaly, pancreatitis

1. *Parotid sialometry* directly measures the quantity of saliva obtained from the parotid duct.
2. In *parotid sialography,* a radiopaque dye is injected into the ductal system. Complications include retention of contrast, which may aggravate local inflammation.
3. *Parotid scintigraphy* capitalizes on the selective ability of the salivary glands to concentrate certain radionuclides and secrete them into the saliva.

These tests can provide a sensitive estimate of salivary gland function but are not specific for Sjögren's syndrome and may be abnormal in other disorders such as sarcoidosis, post-radiation therapy, or diabetes mellitus.

The labial salivary gland biopsy, generally taken from the lower lip, is a simple, well-tolerated procedure. The histopathological features of focal lymphoid infiltration with atrophy of acinar tissue and ductal alterations are highly specific for Sjögren's syndrome. These histologic abnormalities found in the minor salivary gland of the lip are similar to the changes found in the parotid and lacrimal glands and may be used to confirm the diagnosis of Sjögren's syndrome.

Systemic Manifestations

Secretory glands other than lacrimal and salivary glands often are involved *(Table I).* Submucous glands may be involved at all levels of the respiratory tract resulting in nasal dryness, hoarseness, recurrent bronchitis, or pneumonia. Involvement of esophageal mucosal glands, as well as xerostomia, results in dysphagia. The skin is frequently dry, and sweating may be diminished. Vaginal dryness may result in dyspareunia and labial pruritis.

Other systemic manifestations include arthralgias, diffuse myalgia, and Raynaud's phenomenon. A non-erosive arthritis is common. Extra-glandular extension of lymphoid infiltration may involve a variety of organ systems. Kidney involvement may result in renal tubular acidosis or nephrogenic diabetes insipidus. Lymphoid infiltration of the lung may result in chronic interstitial fibrosis. Hepatomegaly and pancreatitis are also seen in Sjögren's syndrome. The most common neurologic involvement is an isolated trigeminal neuropathy, but a peripheral neuropathy may also occur.

TABLE II
SECONDARY SJÖGREN'S SYNDROME — ASSOCIATED AUTOIMMUNE DISEASES

Rheumatoid Arthritis
Systemic Lupus Erythematosus
Scleroderma
Primary Biliary Cirrhosis
Polymyositis
Vasculitis
Thyroiditis
Chronic Active Hepatitis
Mixed Cryoglobulinemia

Rheumatoid Arthritis

Sjögren's syndrome frequently coexists with many other connective tissue diseases *(Table II).* Rheumatoid arthritis is the most common connective tissue disease associated with Sjögren's syndrome. It is found in about 25 per cent of patients with rheumatoid arthritis and correlates directly with its severity and duration. Extraglandular manifestations such as vasculitis, Raynaud's phenomenon, lymphadenopathy, and renal involvement are more common in primary Sjögren's syndrome than in Sjögren's syndrome associated with rheumatoid arthritis.

Laboratory Findings

Laboratory findings commonly found in both primary and secondary Sjögren's syndrome include mild anemia, leukopenia, eosinophilia, elevated erythrocyte sedimentation rate, and diffuse elevation of serum immunoglobulins.

Numerous autoantibodies are found in Sjögren's syndrome *(Table IV).* Rheumatoid factor is the most

TABLE III
SUMMARY OF IMMUNOLOGIC ACTIVITY OF SJÖGREN'S SYNDROME

Prominent lymphocytic infiltration of glandular tissue
Hypergammaglobulinemia
High incidence of autoantibodies
An organ specific (salivary duct) autoantibody
Local synthesis of immunoglobulins
Increased serum and salivary concentration of Beta 2 microglobulin
Presence of circulating immune complexes
Decreased clearance of particulate immune complexes associated with a defect in the Fc receptor function of the reticuloendothelial system

common autoantibody found in Sjögren's syndrome. Antinuclear antibodies are directed against a variety of nuclear antigens in Sjögren's syndrome and may show a homogenous or speckled pattern. Antinative DNA antibodies are rare. Precipitating antibodies to nuclear antigens (SS-A, SS-B) are present in the sera of patients with Sjögren's syndrome. These antigens are immunologically distinct from other nuclear antigens, such as Sm, RNP, or DNA. Antibodies to SS-A and SS-B have been found frequently in the sera of patients with primary Sjögren's syndrome, and less commonly in those with rheumatoid arthritis and Sjögren's syndrome.

Alexander *et al.* (1983) found that antibodies to SS-A and SS-B often were seen in association both with primary Sjögren's syndrome and secondary Sjögren's syndrome with rheumatoid arthritis. Patients with either primary or secondary Sjögren's syndrome, who have antibodies to SS-A, are more likely to develop extraglandular disease. These extraglandular features may include vasculitis, purpura, or lymphadenopathy.

Antibodies to SS-A and, to a lesser extent SS-B, are also found in the sera of patients with SLE but are not found in the sera of patients with other connective tissue diseases. Other autoantibodies found in both primary and secondary Sjögren's syndrome include antisalivary duct, antimitochondrial, antithyroid, and antismooth muscle antibodies *(Table IV)*.

Beta-2-microglobulin is a protein secreted by lymphoid and other cells. It is structurally similar to immunoglobulins. Its function is not known, but it has been found in increased concentrations in the serum and saliva of patients with Sjögren's syndrome. Increased salivary concentrations of Beta-2-microglobulin appear to correlate with the degree of active salivary gland inflammation. The most marked serum elevations are found in those patients

TABLE IV
AUTOANTIBODIES FREQUENTLY SEEN IN SJÖRGEN'S SYNDROME

Rheumatoid Factor
Antinuclear Antibodies
Precipitating Antinuclear Antibodies (SS-A, SS-B, RAP)
Antisalivary Duct Antibodies
Antimitochondrial Antibodies
Antithyroid Microsomal Antibodies
Antismooth Muscle Antibodies

with Sjögren's syndrome, who have associated lymphoproliferation disorders or renal disease. Measurements of serum concentrations of Beta-2-microglobulin may prove useful in identifying those patients with Sjögren's syndrome, who develop serious systemic complications.

Genetic Associations

In addition to clinical and serological differences, there are genetic differences between primary Sjögren's syndrome and Sjögren's syndrome associated with rheumatoid arthritis. HLA-B8 is a histocompatibility antigen that has been associated with a number of autoimmune diseases, including primary Sjögren's syndrome. Another closely linked locus, HLA-D, controls lymphocyte-defined cell surface antigens. In mice, analogues of these loci are closely linked to immune response genes. An increased frequency of the HLA-Dw3 allele has been found in primary Sjögren's syndrome. Rheumatoid arthritis with Sjögren's syndrome is associated with neither HLA-B8 nor HLA-Dw3. Thus, these different genetic markers support the clinical distinction between primary and secondary Sjögren's with rheumatoid arthritis. HLA-B8 is associated with many other autoimmune diseases as well as with Sjögren's syndrome. It may be that HLA-B8 is genetically linked to an immune response gene that predisposes the individual to autoimmune phenomena.

Spectrum of Lymphoproliferation

Clinical and pathologic evidence suggests that a wide spectrum of lymphoproliferation is seen in Sjögren's syndrome, ranging from benign disease with lymphoid infiltrates confined to glandular tissue, to widespread lymphoproliferative malignancy. "Pseudolymphoma" identifies patients in the middle of this spectrum; they have extraglandular extension of non-malignant lymphoproliferation, which

has the potential to regress with therapy or progress to neoplasia.

In the majority of patients with either primary or secondary Sjögren's syndrome, significant lymphoproliferation remains confined to salivary and lacrimal tissue resulting in a chronic, benign course of stable or slowly progressive xerostomia and xeropthalmia. In some patients, however, especially those with antibodies to SS-A, extraglandular extension of lymphoproliferation occurs. The clinical manifestations depend on the organs involved.

Features that may alert the clinician to the possibility of extraglandular disease include regional or generalized lymphadenopathy, hepatosplenomegaly, pulmonary infiltrates, renal insufficiency, purpura, or elevated IgM. A fall in serum IgM is a poor prognostic sign and may herald development of malignant lymphoproliferation, including reticulum cell sarcoma, Waldenstrom's macroglobulinemia, lymphoma, thymoma, or lymphosarcoma.

Treatment

Patients with keratoconjunctivitis sicca should be followed by an ophthalmologist to detect and treat early ocular complications. Local symptomatic improvement may be obtained by methylcellulose eye drops. Oral symptoms or xerostomia may be treated with saliva substitutes, some of which contain methylcellulose. Careful dental hygiene and frequent dental examinations may prevent dental complications. Bromhexine, a drug that increases bronchial secretions and reduces viscosity, has been used in Europe for treatment of chronic bronchitis. This drug has been reported to improve lacrimal and salivary secretions also.

Corticosteroids and immunosuppressive agents have been reported to be beneficial in severe cases of primary Sjögren's syndrome, but randomized controlled trial data are not available. Since the disease is usually benign and chronic, the side effects of these drugs usually outweigh the benefits. Extraglandular extension, or pseudolymphoma, may be treated with radiation, corticosteroids, or immunosuppressive agents, depending on the organs involved.

Summary

Sjögren's syndrome is a chronic inflammatory autoimmune disorder characterized by a triad of keratoconjunctivitis sicca, xerostomia, and connective tissue disease. Diagnosis may be made with only two of the three major features of the triad. Cases of xerophthalmia should have an ophthalmologic examination with Schirmer's test, rose bengal and flourescein staining, and slit lamp evaluation to establish the presence of keratoconjunctivitis sicca. Additional studies, such as labial salivary gland biopsy and serologic testing for antinuclear antibodies, are not routinely necessary but may be helpful to confirm the diagnosis in questionable cases, especially those with extraglandular involvement. Treatment of sicca symptoms should be conservative since the usual course is benign and mild. Treatment with immunosuppressive agents and corticosteroids should be reserved for severe extraglandular involvement, pseudolymphoma, and lymphoreticular malignancies. Sjögren's syndrome has evolved from a medical curiosity to a disorder that overlaps a number of areas of major interest — the lymphoproliferative malignancies, the autoimmune disorders, and the dysproteinemias. Further study of this syndrome offers great potential for elucidating the interrelationships among these areas.

Self-Assessment Questions

Directions: Choose the single best answer.

1. Which test is most specific for oral involvement in Sjögren's syndrome?
 a. Parotid sialography
 b. Parotid scintiscanning
 c. Parotid flow measurements
 d. Labial salivary gland biopsy
2. Which of the following is (are) more commonly found in association with primary Sjögren's syndrome as compared with secondary Sjögren's syndrome with rheumatoid arthritis?
 a. Antibodies to SS-A, SS-B
 b. HLA-B8
 c. Extraglandular manifestations
 d. All of the above
3. Which of the following suggests extraglandular involvement in Sjögren's syndrome?
 a. Hepatosplenomegaly
 b. Pulmonary infiltrates
 c. Renal insufficiency
 d. All of the above
4. A fall in total serum IgM is a poor prognostic sign and may herald the development of malignant lymphoproliferation. (T or F)
5. Symptoms of xerostomia and xeropthalmia are best treated with systemic corticosteroids. (T or F)

(Answers on page 503)



Drug INFORMATION

JOYCE GENERALI, R.Ph., *Editor*

An Update on Insulin

Question: There have been several new developments in the manufacturing and marketing of insulin. Will you provide an update on the insulins currently available in the United States?

Answer: Purity and species (source) of insulin are two important factors in its clinical use because each plays a role in causing insulin side effects. These include injection site lipoatrophy or hypertrophy, insulin allergy, and insulin antibody formation with or without insulin resistance.[1, 2]

Since 1972, technological advances have allowed manufacturers to produce highly purified insulin products — *i.e.* ≤ 10 parts per million (ppm) of proinsulin contamination. The most important factor in antigenicity, however, is the animal source from which the insulin is derived.[3-5] The newer, more purified insulins are produced from pork which differs from human insulin by only one amino acid.[1] Insulin from beef sources differs from human insulin by three amino acids and is more antigenic than that from pork sources.[4] Many diabetic patients will develop antibodies to conventional (>10,000 ppm) or the single peak (300-3,000 ppm) insulins.[5-7] Patients who are transfered from conventional to highly purified (≤10 ppm) insulins have a marked decrease in insulin antibody levels.[3, 4] The role of insulin antibodies, however, has not been clarified.

(Continued on page 502)

TABLE I

INSULINS CURRENTLY AVAILABLE IN THE U. S.

Purity	*Specie Source*	*Time of Action* RAPID ACTING	INTERMEDIATE ACTING	LONG ACTING
<10 ppm*	Pork	Iletin II-Regular (Lilly) Actrapid (Regular) (Squibb-Novo) Semitard (Squibb-Novo) Insulin Quick, Velosulin (Nordisk) Mixtard — 30%	Iletin II-NPH (Lilly) Iletin II-Lente (Lilly) Monotard-Zinc (Squibb-Novo) Insulatard-NPH (Nordisk) Protaphane-NPH (Squibb-Novo) Regular/70% NPH (Nordisk)	Iletin II-PZI (Lilly)
	Beef	Iletin II-Regular (Lilly)	Iletin II-NPH (Lilly) Iletin II-Lente (Lilly)	Iletin II-PZI (Lilly) Ultratard (Squibb-Novo)
	Beef/Pork		Lentard (Squibb-Novo)	
< 20 ppm	Beef/Pork	Iletin I-Regular (Lilly) Iletin I-Semilente (Lilly)	Iletin I-NPH (Lilly) Iletin I-Lente (Lilly)	Iletin I-PZI (Lilly) Iletin I-Ultralente (Lilly)
< 25 ppm	Pork	Regular (Squibb-Novo)		
	Beef	Semilente (Squibb-Novo)	Lente (Squibb-Novo) NPH (Squibb-Novo)	Ultralente (Squibb-Novo) PZI (Squibb-Novo)

Drug Information Service, Joyce Generali 6/83

* Internal specifications for manufactures may vary

The President's Message



This special issue marks the tenth consecutive year that the *Journal* has focused on The University of Kansas School of Medicine-Witchita.

From its inception, UKSM-W has grown and matured into a national model for community based clinical training. A progress report can be found in the Dean's Letter elsewhere in the *Journal*.

The stature of the Wichita program is a testimonial to the vision, cooperation, and hard work of many people. They include:

- Administrators in Wichita and Kansas City who conceived and implemented the program;
- State officials who allocated funds;
- The citizens of Wichita who have integrated the institution into the overall community structure;
- The local hospitals — St. Joseph, St. Francis, Wesley, and the V.A. — whose facilities and personnel have provided the essential clinical setting;
- The students who have elected to pursue their clinical experience within the Wichita program;
- The staff and faculty who have dared to execute innovative policies;
- The medical community in Sedgwick and surrounding counties who have made an immeasurable contribution to medical education in Kansas through volunteer service to and support of the program.

The Kansas Medical Society salutes The University of Kansas School of Medicine-Wichita and those who have contributed to its growth and development.

James A. Gleason M.D.

President



Medical Costs: The Prodigal Sum

One of the more frequently heard medical terms of the day — close behind herpes and AIDS — is cost containment. This means "economizing" but avoids some of the disagreeable directness and sense of futility associated with the ordinary term. By whatever name, medicine can't claim exclusive rights to it. The subject has recurred in one guise or another at frequent intervals ever since Eden Enterprises went out of business because the operators misunderstood its relationship to the law of supply and demand.

"Cost containment" combines generality and specificity and at this point is still to be considered prospective. It has yet to arrive in substantial form but the impending birthing has brought forth a variety of midwives eager for the honor of bringing the recalcitrant infant into our midst. Different though the proposed methods are, they have the common distinction of trying to regulate someone else's behavior. The preparatory activity has brought with it a variety of acronyms — HMO, DRG, PPO, and so on. One gets the feeling that our world (medical division, at least) is the object of an intensive genetic engineering experiment in which these triplet codons are to be insinuated into the social DNA with the hope of some marvelous medical mutation.

Social and political activists, not always so fervent in pursuit of restraints, are promoting their plans with conviction born not only of righteous certainty but of the knowledge that there is enough discontent at any given moment to provide support for something different. As a matter of fact, the elements of our current problems are so complex and catholic that any plan will probably do something and none will do everything. In the long view, they are expedients occupying our attention while some undetected process of social osmosis is producing the real changes. Despite the inclusion of physician services directly or indirectly in most of the plans, the medical profession may well feel itself increasingly isolated from the process. Hospitals, in defending their extended turf, are nullifying their relationship with physicians either by making them employees or citing fiscal justification for regulating their services. Blue Cross and Blue Shield, developed by physicians and hospitals to meet the same problems in another era, have obtained a divorce although not without some problems of custody of the children. The government, insurance companies, and business are imposing extended requirements on behalf of citizens, clients, and employees and, in saying what will and will not be provided and how, they are, in effect, telling physicians what they can and cannot do. In the hurly-burly of the struggle, the understanding is all but lost that such pressures are often dividing physicians from each other as well as from their once and final allies, their patients.

So it is that this proliferation of plans for resolving the problems of health care costs plays counterpoint to the Greek tragedy chorus in the background chanting its doleful lament that they are too high. The proposals being invoked currently take predictably different forms according to inspiration from some presumed defect in the medical scene which, it is felt, will be corrected by the proposed plan. Bookkeeping requirements have, for example, long imposed various systems of categorizing illnesses and consequently the payment for their management. This has come to a sort of fruition in the DRG system in which it is no longer a bookkeeping convenience but a straightforward dictation of how much will be paid for what according not necessarily to the care given but the name of the condition. It is hoped that patients will be as well disciplined in the course of their illnesses.

Since physicians' fees are one of the more evident contributions to the total health bill, they have come in for their fair share of revisionist attention — and more. The most direct approach has been based on the tacit acceptance that physicians should receive something but those who charge the least are pre-

ferred. They will be given a seal of approval entitling them to referrals (economic basis, not professional) and it is anticipated that the system will inspire physicians to compete in reducing their charges (encouraged by a reported oversupply of physicians as well as a public already indoctrinated to comparison shopping and advertising inserts of varying hue). For the public, there will be the obvious effect of pitting providers against each other in the fight for preference, with the double benefit of obtaining the lowest price and disrupting the cohesion of the physician group.

From another direction, the HMO philosophy expresses the belief that economy in the medical market will be accomplished if everyone will stay healthy. This will entail some changes in personal habits, industrial methods, the weather, and across-the-board mores, to touch on only a few factors. The plan obviously isn't without its problems but we are assured that the prospect of saving some money will be sufficient motivation for the citizenry (the employer segment, anyway) and the health sellers are in full cry. Such a plan carried to a logical extreme might well lose physician support since its ultimate success would seem to eliminate physicians altogether.

Our reading of the current medical-economic contention is not quite as choleric as these words may imply. However, our acknowledged paranoia — as we have indicated in the past — sees a degree of societal involvement in which medical control is being progressively withdrawn from medical practitioners through the economic pragmatism of fiscal control. It is hard to imagine any approach that has not been advocated in recent years and our purpose is not to produce another one but to attempt a degree of perspective which will be essential to the development of any viable system. Our complaint, essentially, is that the planners, in their zeal to achieve the blessings of cost effectiveness and, at the same time, care for all, fail to consider certain unique features of medical care which must be taken into account before a stable, equitable, productive mechanism for medical service can be maintained — something that, to the extent we have had it in the past, has been the contribution of the medical profession more than any other segment in society.

One of the more basic features of medical care is the fact that the "product" resulting from its function is intangible. The input — services of personnel at all levels, equipment, facilities, medications — are compatible with the laws economists like to apply in measuring tangible values. But the medical "industry" does not produce tons of goods or measurable quantities of substance that can be given a direct monetary value. The end product is the relief of the affliction at whatever level and its measure is the removal of the patient from the medically dependent state and return to productivity — and it has been pointed out frequently that the survival of the disabled (whether by age or physical or mental status) is a serious disruption to conformity to economic rules. Periodically, one or another bureau issues figures purporting to measure the cost to the national economy of various illnesses. Whether valid or not, these are before the fact. What is the worth to the national economy of the return of the patient to work and can, in total, it ever repay what was lost during the individual's absence? What part of our national productiveness (read wealth) can be attributed to the effectiveness of this medical service? If health care costs too much, in relation to what? There has been much talk of "cost effectiveness" related to some specific item or activity but how do we arrive at the cost effectiveness without knowing what that effectiveness is?

At the same time, there is the matter of intrinsic effect — the admitted inherent prodigality of medical care. Here medical care definitely departs from any effort to fit into the commercial mold. There should be (and without the pressures of bureaucracy, usually is) one level of medical service — the best possible within limits of specifics. A given physician may not be the wisest product of medical academia, the facilities may not be total and superb, numerous factors may keep "best" from being "ideal," but we submit that no service is more consistently motivated by the intent to provide first quality to all than medicine. There was a time when medical service, both philosophically and practically, was provided only to those of relative wealth. The poor doctored themselves — or might be provided with domiciliary care, mainly when it was deemed safer or pleasanter by the well-to-do to so segregate them. It has been only recently (in the long view) that society has adopted the concept of medical care for all and, happy enough to accept the implied virtue, is still having trouble with the matter of cost.

Compounding this problem, the development of high technology assures that an uneconomical system will be in effect from now on — unless society deliberately chooses to put a moratorium on medical progress (a certain impossibility). Even before any new technologic development can be put on an economically feasible basis, the process has moved on (with public pressure) to the next level of achieve-

(Continued on page 501)

Pestilence in Ahs

Rocky Mountain Spotted Fever in Kansas

G. William Nice, M.D., *Topeka*

Incidence

Rocky Mountain spotted fever is a mild to very severe disease first described in the Rocky Mountain area in 1896. It now occurs in 46 states as well as Canada, Mexico, and Central and South America. The main insect vectors include the wood, dog, rabbit, hard, and brown ticks. The appropriate technique for tick removal is illustrated in *Figure 1*. The ticks are frequently transmitted by dogs, but these organisms may be maintained in rabbits and other small mammals. The disease may rarely be transmitted from person to person by infected respiratory droplets, especially if there is bronchitis with sputum-producing cough. Mortality rates range from 5 to 20 per cent.

Pathology

The characteristic pathological lesion is a vasculitis and occurs in the small blood vessels in the skin, subcutaneous tissue, central nervous system, lungs, heart, kidneys, liver, and spleen.

Clinical Course

There is a history of tick infestation in about 70 per cent of cases — usually in persons who are out-of-doors such as farmers, foresters, surveyors, road crews, cattle and sheep herders, and campers. It occurs more commonly from May to September in the northern states; however, the incidence is fairly constant during the entire year in the southern states. The longer the tick bites, the greater the chance of infection to the host. The incubation period varies from 3 to 12 days, but averages about seven days from the time of the tick bite. As a general rule, the disease is more severe when the incubation period is short (3-5 days). Onset is usually rapid with severe headache, fever, muscular pain, and a feeling of severe prostration. Other symptoms may include nausea, vomiting, edema, conjunctival infection, photophobia, abdominal pain, and arthralgia. Temperature usually remains elevated for several days after improvement has begun.

Address reprint requests to Dr. Nice, 112 Medical Arts Building East, Topeka KS 66604.

The characteristic rash usually begins two to six days after the original symptoms of fever, chills, headache, and muscular aching. The rash usually starts on the wrists and ankles and then rapidly spreads over the extremities, trunk, and head in a few hours to one or two days. The rash will frequently improve when the fever disappears, but may remain as pigmented spots for several weeks.

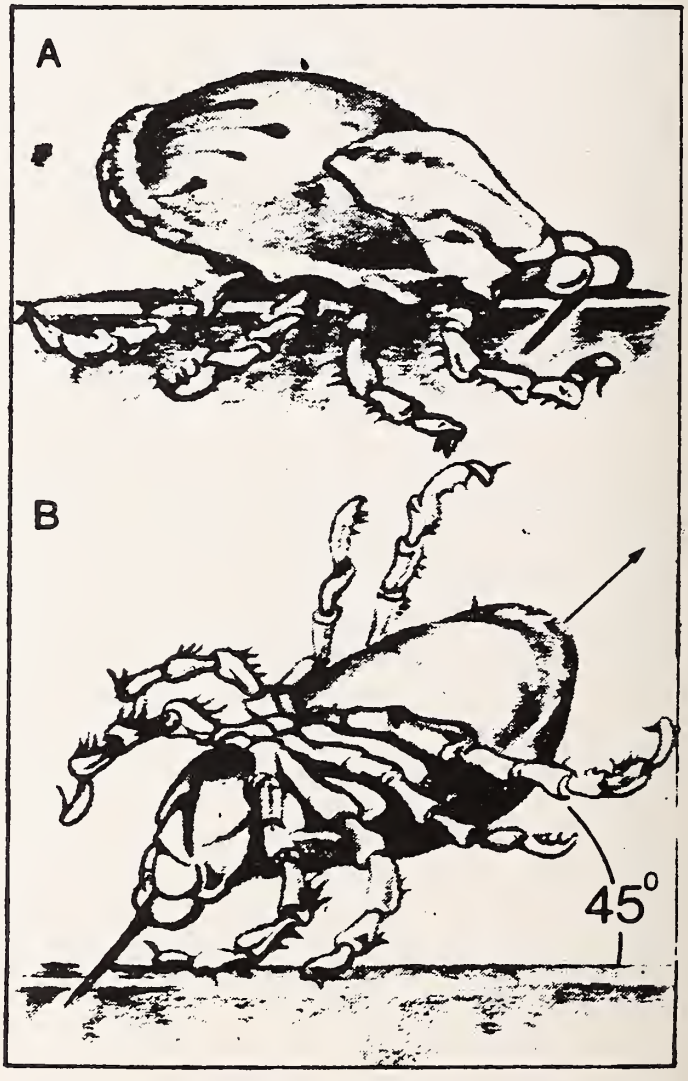


Figure 1. To remove a tick, DO NOT attempt to pull its body straight out. The tick's mouthpiece points caudad (A) and will break off, leaving the head and mouthpiece still imbedded. Flip the tick over (B) and pull steadily with tweezers at a 45-degree angle.

Neurologic symptoms such as severe headache, deafness, central blindness, hyperesthesia, ankle clonus, restlessness, insomnia, delirium, and coma indicate the presence of encephalitis. If the patient recovers, learning disabilities frequently occur.[1]

In the more severe cases, there may be cardiac involvement with prolonged P-R interval and also ST changes on the electrocardiogram. Hypotension may reach shock levels with circulatory failure. Cardiac arrest may occur in the more severe cases — usually one to two weeks after onset. Three important findings include vasculitis, thrombocytopenia, and disseminated intravascular coagulation; if present, these require immediate intensive therapy.[1] Persistent abdominal pain, constipation, and distention frequently indicate a fatal outcome. Secondary infections such as pneumonia, otitis media, and parotitis may occur. Convalescence is slow; complete recovery may take weeks, months, or even a year.

Untreated Rocky Mountain spotted fever will improve in two or three weeks in mild cases and three to four weeks in moderately severe cases. Severe cases may develop early onset of deep coma, high fever, signs of respiratory and cardiac distress, shock, thrombocytopenia, hypofibrinogenemia, or severe purpura which require immediate treatment. Other serious developments may include circulatory failure, secondary hemorrhage in the lungs or brain, jaundice due to liver damage, and arterial thrombosis producing necrotic gangrene of fingers, toes, earlobes, or scrotum. A rash may appear later or not at all.[2, 3]

Fulminant disease may cause death on or before the fifth day.[4] These cases usually have extensive central nervous system involvement and frequently do not develop a rash.

Laboratory Tests

The complement fixation agglutination or neutralization tests are more reliable than the Weil-Felix reaction and should be used to confirm the diagnosis. Complement fixation antibodies usually appear during the second and third weeks. It is unusual for them to appear before the 14th day of illness, and occasionally a rise in the titer may not occur until a month or more after the onset of the disease. In patients who receive specific antibiotic therapy, the antibodies may be delayed; therefore, specimens should be checked at intervals of two and six weeks later.

Other diagnostic laboratory studies include a microagglutination technique employing specific antigen and immunofluorescent techniques to detect R. rickettsia in ticks, chick embryo, and tissues of animals; lymphocytic blastogenic responsiveness to rickettsial antigens has been demonstrated. Skin biopsy of the rash can demonstrate the disease as early as four hours after the biopsy and as early as the third or fourth day of the illness.

Other laboratory studies should include complete blood count for leukopenia, platelet count, electrocardiogram, liver function test if liver disease is suspected, chest x-ray if secondary pneumonia is suspected, electroencephalogram if encephalitis is suspected, and electrolyte studies in moderate or severe cases. Blood urea nitrogen and creatinine must be checked if renal damage is suspected, and prothrombin time and partial thromboplastin studies are useful in treating disseminated intravascular coagulation.

Treatment

Tetracycline and chloromycetin are the drugs of choice, and most prefer tetracycline in a daily dose of 25-50 mg/kg of body weight. Usually 2 gm/day in divided doses will be adequate for most adults. Intravenous tetracycline should be given in divided doses of 0.5 gm/6-12 hrs. Up to 0.5 gm of tetracycline may be given intramuscularly. Chloramphenicol is given in doses of 50-75 mg/kg of body weight in adults or children. Sulfonamides should not be given because they usually make the disease worse. Steroids may be useful in conjunction with the antibiotics, especially in the patient with neurological disease.

Management of liver and renal disease is directed at the specific abnormality in conjunction with antibiotic therapy. Pulmonary edema and congestive heart failure will require oxygen, diuretics, lanoxin, and close followup of renal disease.

Gangrene, decubitus ulcers, and thrombophlebitis should be treated by the surgical and medical treatment program.

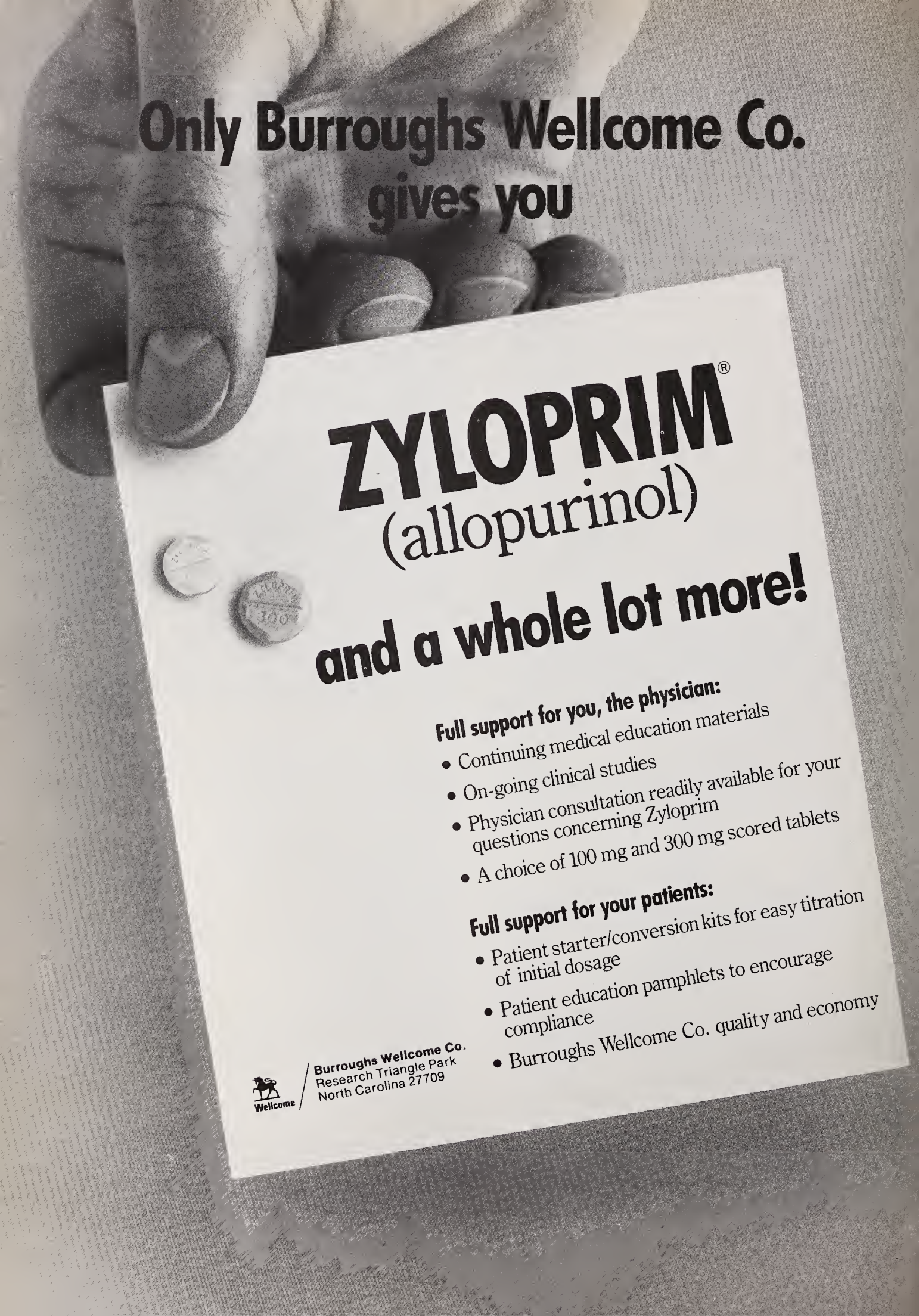
Good nursing care is a necessary part of treatment, especially in the severely ill or comatose patient. This should include careful cardiac and neurological monitoring as well as special skin care and frequent turning of the comatose patient to prevent decubitus ulcers, pneumonia, and thrombophlebitis.

Antibiotic therapy given at the time of the tick bite does not prevent Rocky Mountain spotted fever, but may delay the onset.

Summary

When the patient has a known history of tick bite with a rapid onset of headache, fever, chills, muscle aching, and especially with a rash, treatment with

(Continued on page 493)


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Pestilence in Ahs

(Continued from page 491)

TABLE I
ROCKY MOUNTAIN SPOTTED FEVER IN KANSAS — 1970-1982

Year	*# of cases*	*Year*	*# of cases*
1970	1	1977	11
1971	4	1978	7
1972	2	1979	8
1973	6	1980	11
1974	6	1981	14
1975	10	1982	11
1976	9		

tetracycline should be started promptly and the patient should be observed closely for at least two to four days. Confirmed cases of Rocky Mountain spotted fever in Kansas as reported by the Kansas Department of Health and Environment for the years 1970-1982 are shown in *Table I*.

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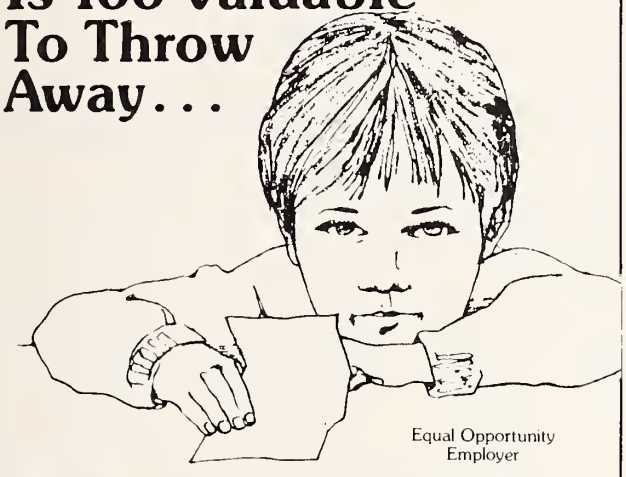
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Mannitol Intoxication

(Continued from page 460)

have been dangerous and could have precipitated pulmonary edema. Because of the high extracellular fluid osmolality due to mannitol, infused fluids would not be expected to enter cells where osmolality would be lower. Infused fluid could not be excreted by the kidney because of the acute renal failure. As noted, several functional parameters indicated a component of acute renal failure in addition to pre-renal disease resulting from a decrease in renal blood flow due to the osmotic diuresis. The extrarenal clearance of mannitol is very slow in patients with renal failure. Extrarenal metabolism includes conversion to other compounds and excretion in bile.

Using the osmolality gap as an indirect measurement of mannitol concentration it has been found that hemodialysis results in a mannitol half-life of 6 hours, compared to a half-life of 36 hours with no dialysis treatment. Mannitol half-life with peritoneal dialysis is 21 hours.[3] Hemodialysis was undertaken in this patient for several reasons, including:

1. The large dose of mannitol received by the patient as determined from nursing records, and reflected in the large calculated osmolality gap.
2. Significant mental obtundation and hypoventilation.
3. Hyperkalemia.
4. Oliguric renal failure precluding safe intravenous fluid administration.

The patient underwent three daily four-hour hemodialysis treatment periods using the subclavian vein for vascular access. Removal of mannitol with hemodialysis permitted repletion of fluid and electrolytes without fear of precipitating pulmonary edema. With hemodialysis removal of mannitol and replacement of fluid, the urine output gradually improved during the next several days with re-establishment of renal function. The impacted right ureteral calculus was surgically removed through a flank incision three days after discovery of the mannitol infusion. The post-operative course was uneventful and the patient was discharged on the 11th hospital day. At the time of discharge, serum creatinine was 1 mg/dl and blood urea nitrogen, 13 mg/dl.

The patient's renal stone was found to be composed of calcium oxalate monohydrate. Subsequent examinations identified the presence of absorptive hypercalciuria associated with hyperuricosuria. The patient is presently being treated for these abnormalities.

Summary

An unusual case history of mannitol intoxication has been presented and discussed. Mannitol intoxication is rare and usually occurs in a setting of pre-existing renal failure. The pre-eminent clinical finding is central nervous system depression with lethargy and mental obtundation. In addition to the examination of routine serum electrolytes, it is helpful to calculate the osmolality gap, which is the difference between measured and calculated serum osmolality. This value serves as an indirect measurement of the serum mannitol level, and can help guide therapy. Hemodialysis is the most efficient means of lowering the serum osmolality through removal of mannitol and should be employed in severe cases such as was seen in this patient.

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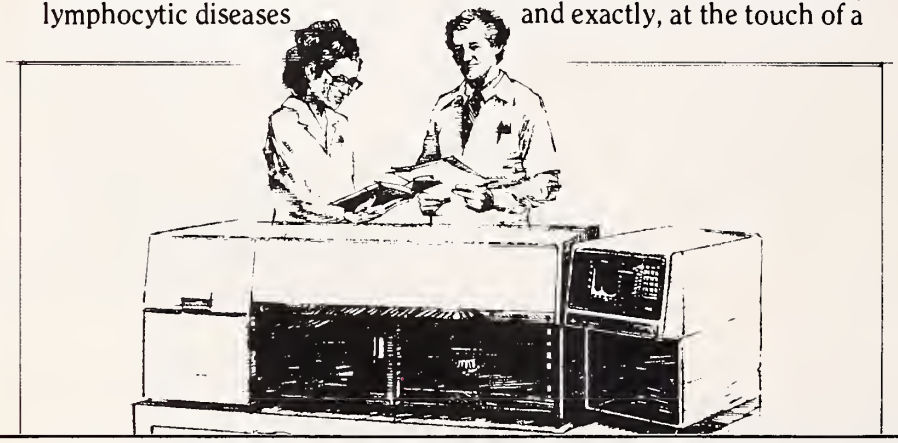
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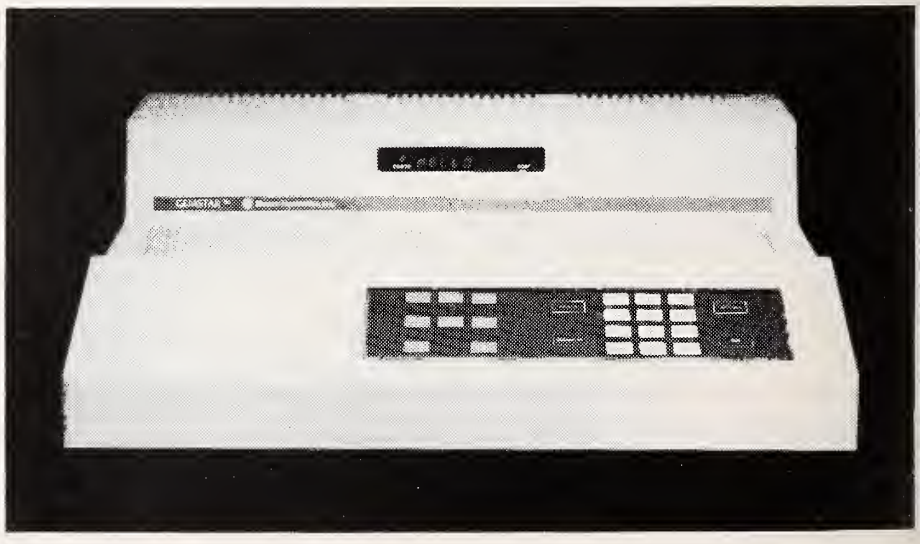


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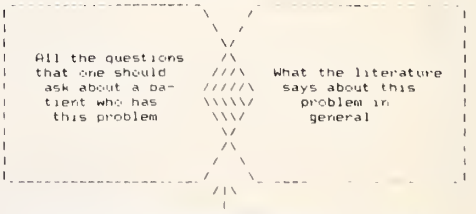
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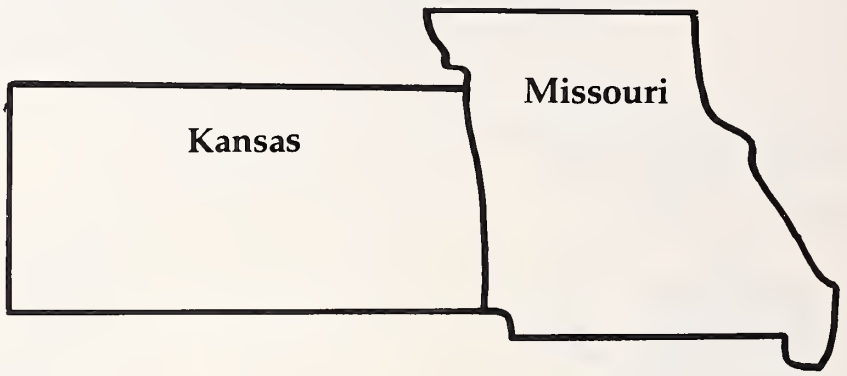
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(Continued from page 474)

available on a regular basis, ability to provide thorough training of these individuals, and a nursing support system to provide the necessary followup.

Acknowledgements

The Kansas Department of Health and Environment provided block grant funds to UKSM-W to support hypertension screening services to Sedgwick County residents.

Individuals and sponsoring agencies instrumental in establishing the fire station screening program include: Eva Benson, R.N., American Heart Association; Chief Jim Cloud, Wichita Fire Department; Marilyn Crowley, R.N., CCRN, EMICT, Wichita Fire Department; RoxAnn Dicker, R.N., M.N., UKSM-W; Terry Kloppenberg, International Order of Firefighters, Local 666; Tom Maher, Ph.D., American Heart Association; Nancy Monnat, UKSM-W; and Linda Wright, American Heart Association.

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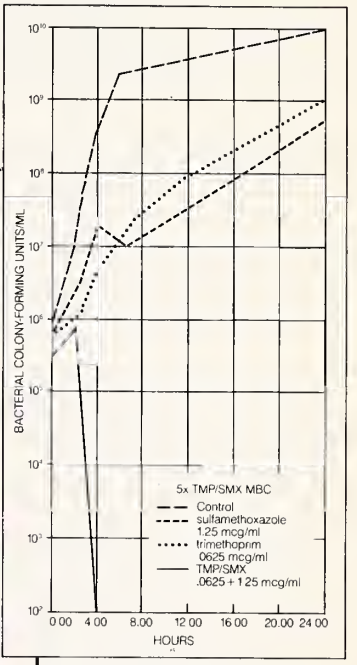
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Kill curve kinetics of Bactrim and its individual components against *E. coli in vitro*.[1]

The bactericidal action of Bactrim has been demonstrated *in vitro* on laboratory strains of *E. coli*[1,2] and on clinical isolates of *E. coli, Klebsiella-Enterobacter, Proteus mirabilis* and *Morganella morganii*[3]—the most common causative organisms of urinary tract infections.[4] More than 100 published studies attest to the efficacy of Bactrim in recurrent urinary tract infections due to these organisms.[5] In comparative studies with other antimicrobials, Bactrim has consistently demonstrated unsurpassed efficacy during therapy.[6-11]

Resistance to Bactrim develops more slowly than to either of its components alone *in vitro*.* Among urinary tract isolates, resistance has rarely emerged in susceptible strains.[5,12] Bactrim is contraindicated in pregnancy at term, during lactation, in infants less than two months old and in documented megaloblastic anemia due to folate deficiency.

Initial episodes of uncomplicated urinary infections should be treated with a single-agent antimicrobial.

Bactrim™ DS

(trimethoprim and sulfamethoxazole/Roche)

b.i.d. *for recurrent urinary tract infections*

**In vitro* data do not necessarily predict clinical results.

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See next page for references and a summary of product information.

References: 1. Data on file, Hoffmann-La Roche Inc., Nutley, NJ. 2. Kramer MJ, Mauriz YR, Robertson TL, Timmes MD: Morphological studies on the effect of subinhibitory and inhibitory doses of sulfamethoxazole-trimethoprim combination on *Escherichia coli*. Presented at the 12th International Congress of Chemotherapy, Florence, Italy, Jul 19-24, 1981. 3. Spicehandler J *et al: Rev Infect Dis* 4:562-565, Mar-Apr 1982. 4. Stamey TA: *Pathogenesis and Treatment of Urinary Tract Infections*. Baltimore, Williams & Wilkins, 1980, p. 13. 5. Ronald AR: *Clin Ther* 3:176-189, Mar 1980. 6. Cooper J, Brumfitt W, Hamilton-Miller JMT: *J Antimicrob Chemother* 6:231-239, 1980. 7. Gower PE, Tasker PRW: *Br Med J* 1:684-686, Mar 20, 1976. 8. Cosgrove MD, Morrow JW: *J Urol* 111:670-672, May 1974. 9. Iravani A *et al: Antimicrob Agents Chemother* 19:598-604, Apr 1981. 10. Schaeffer AJ, Flynn S, Jones J: *J Urol* 125:825-827, Jun 1981. 11. Rous SN: *J Urol* 125:228-229, Feb 1981. 12. BAC-DATA Medical Information Systems, Inc., Bacteriologic Reports, Winter Series, 1976-82.

Bactrim™ DS

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii*. It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, hepatocellular necrosis, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients. *Pregnancy:* Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, hepatocellular necrosis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: *Not recommended for infants less than two months of age.*

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:

Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS:

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 20. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. *Pediatric Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz (1 pint). *Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per tea spoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).

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References:
1. Stone PH, Turi ZG, Muller JE: Efficacy of nifedipine therapy for refractory angina pectoris. *Am Heart J* 104:672-681, September 1982.
2. Antman E, Muller J, Goldberg S, et al: Nifedipine therapy for coronary-artery spasm: Experience in 127 patients. *N Engl J Med* 302:1269-1273, June 5, 1980.

BRIEF SUMMARY

PROCARDIA® (nifedipine) **CAPSULES** For Oral Use

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria. 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents: (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PRDCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

More detailed professional information available on request

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"I can do things that I couldn't do for 3 yrs. including joining the human race again."



Quotes from an unsolicited letter received by Pfizer from an angina patient. While this patient's experience is representative of many unsolicited comments received, not all patients will respond to Procardia nor will they all respond to the same degree.

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"My daily routine consisted of sitting in my chair trying to stay alive."

"My doctor switched me to PROCARDIA[*] *as soon as it became available. The change in my condition is remarkable."*

"I shop, cook and can plant flowers again."

"I have been able to do volunteer work...and feel needed and useful once again."

PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks,[1] taking fewer nitroglycerin tablets,[2] doing more, and being more productive once again.

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%).



for the varied faces of angina

* Procardia is indicated for the management of:
1) Confirmed vasospastic angina.
2) Angina where the clinical presentation suggests a possible vasospastic component.
3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

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Please see PROCARDIA *brief summary on adjoining page*

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J-9043-4 July 1981

Medical Costs: The Prodigal Sum

(Continued from page 489)

ment, and the medical service presents a bill that can never be justified in standard economic terms. Moreover, the potential buyer in the general market place can choose, according to his desires, needs, and ability to pay, from a number of types and qualities of a given product — or choose not to buy. But the "consumer" of the medical "product" rarely has that degree of choice. Not only is the need for care established without the patient's intention or permission (aside from those grossly aberrant habits that recognizably produce damage) but the care (and its cost) are more rigidly prescribed, again without the opportunity for the patient to select the style and form most appealing. There is, indeed, room for economy in the over-all conduct of medical service and although physicians will be on the cutting edge (pun or not, as you prefer), adjustments will be required at all levels. We have, for example, obviously and deliberately ignored the matter of inflation and its role in our current difficulties. This is an extensive and tragic phenomenon operating in all areas.

No plan will work better than its lowest level of effect, and the total value is more in the maintenance of awareness than in any specific contribution. Physicians will be vitally affected since they are ultimately the ones who must accommodate the inequities between the economic realities of materialistic principles and the medical realities of health support. The public still has trouble relating these unique features of medicine to their concept of economics — until they become sick. *Then* is the time to discuss cost containment. — *D.E.G.*

Erectile Dysfunction

(Continued from page 469)

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Drug Information

(Continued from page 486)

Indications for the purified insulins may include local or systemic allergy, insulin resistance, or lipoatrophy. Patients receiving intermittent or short term insulin therapy — *i.e.*, surgery, pregnancy, infection — may also benefit from the purified insulin products.[8-10]

In the past year, there have been several new developments in the manufacturing and marketing of insulin which led to some confusion for both physicians and patients. Squibb-Novo, Inc. was organized in 1982 and is jointly owned by Squibb and Novo Industries A/S. Although insulin labeling changes have resulted, no change was made in the insulin itself or to the manufacturing procedures or testing. Squibb-Novo, Inc. now manufactures single peak (<25 ppm) insulins (previously Squibb) and purified insulins (previously Novo).[11]

Also, Eli Lilly has increased the purity of its beef/pork insulin to contain less than 20 ppm of proinsulin and other non-insulin proteins. Currently, there are no insulin products available in the United States containing more than 25 ppm of proinsulin. The clinical significance between 20 ppm *vs* 25 ppm contamination is probably negligible. The major difference is the pork *vs* pork-beef species source. Insulins currently available in the United States are listed in *Table I*.

Humulin, the Lilly brand of biosynthetic insulin of recombinant DNA origin should be available in mid 1983.[12] This manufacturing process provides insulin that is identical in amino acid sequence and structure to human insulin and is independent of the future supply of pancreas glands.[13] The indications for using human insulin are the same as those for the purified pork insulins.

Submitted by
M. Annette Cordova, R.Ph.

References are available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

Sjögren's Syndrome

(Continued from page 485)

Answers

1. d
2. d
3. d
4. *True*
5. *False*

Suggested Readings

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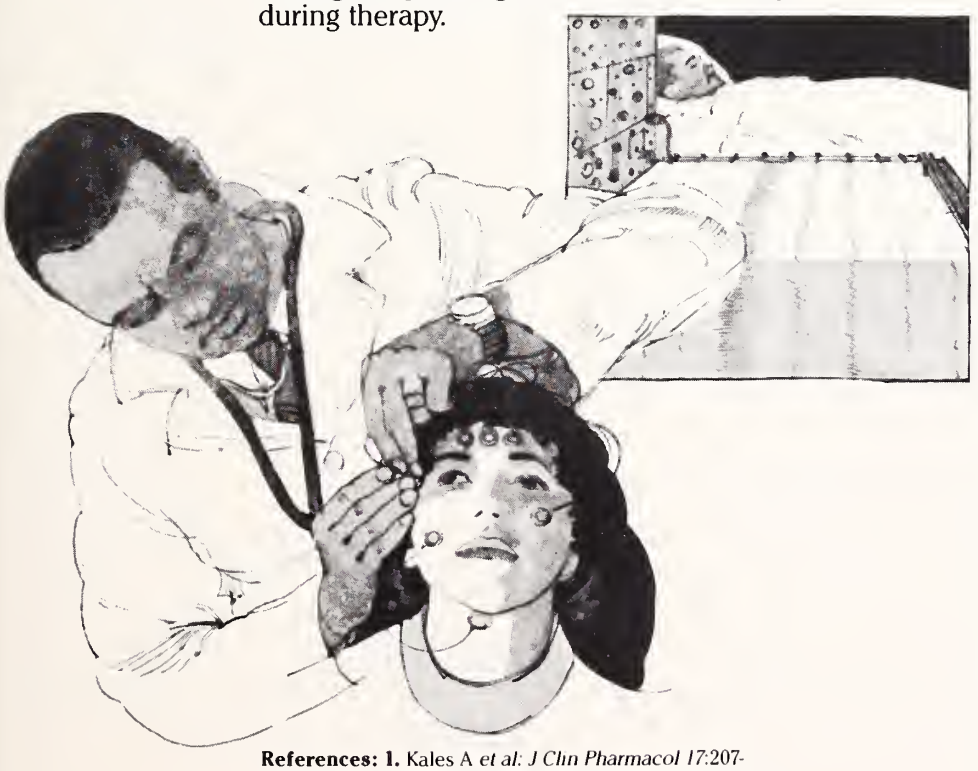
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- Studied extensively in the sleep laboratory—the most valid environment for measuring hypnotic efficacy.[1-12]
- Studied in over 200 clinical trials involving over 10,000 patients.[13]
- During long-term therapy, which is seldom required, periodic blood, kidney and liver function tests should be performed.
- Contraindicated in patients who are pregnant or hypersensitive to flurazepam.
- Caution patients about drinking alcohol, driving or operating hazardous machinery during therapy.



References: 1. Kales A *et al: J Clin Pharmacol 17*:207-213, Apr 1977 and data on file, Hoffmann-La Roche Inc., Nutley, NJ. **2.** Kales A: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **3.** Zimmerman AM: *Curr Ther Res 13*:18-22, Jan 1971. **4.** Kales A *et al: JAMA 241*:1692-1695, Apr 20, 1979. **5.** Kales A, Scharf MB, Kales JD: *Science 201*:1039-1041, Sep 15, 1978. **6.** Kales A *et al: Clin Pharmacol Ther 19*:576-583, May 1976. **7.** Kales A, Kales JD: *Pharmacol Physicians 4*:1-6, Sep 1970. **8.** Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc 27*:541-546, Dec 1979. **9.** Dement WC *et al: Behav Med 5*:25-31, Oct 1978. **10.** Vogel GW: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **11.** Karacan I, Williams RL, Smith JR: The sleep laboratory in the investigation of sleep and sleep disturbances. Scientific exhibit at the 124th annual meeting of the American Psychiatric Association, Washington, DC, May 3-7, 1971. **12.** Pollak CP, McGregor PA, Weitzman ED: The effects of flurazepam on daytime sleep after acute sleep-wake cycle reversal. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, June 30-July 4, 1975. **13.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ.

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Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended. Repeated therapy should only be undertaken with appropriate patient evaluation.

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Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

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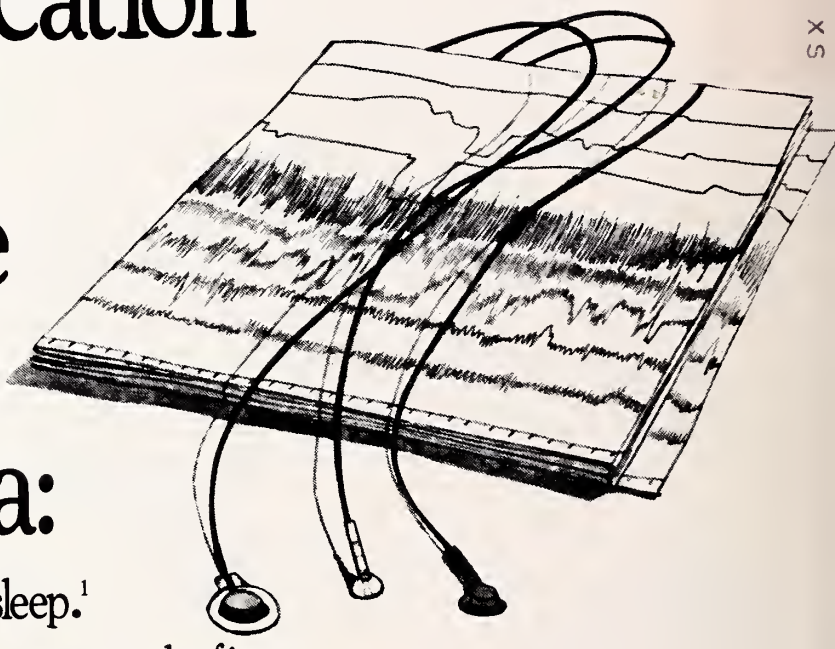
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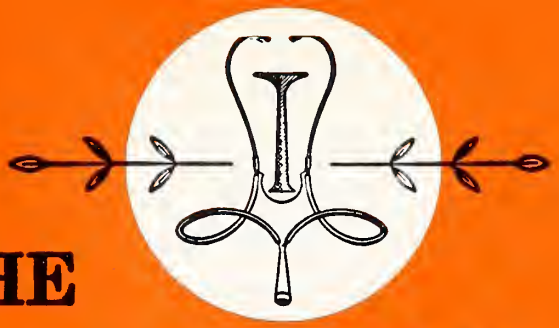
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Book REVIEWS

Getting Them Sober, by Toby Rice Drews. Haven Books, Division of Logos International, copyright 1980. 205 pages.

Author Toby Rice Drews, M.L.A., is a clinician, a licensed social worker/counselor who works actively with alcoholic families. She is also professor of counseling at Johns Hopkins University and a free lance writer and editor who was raised in an alcoholic family.

Have you ever wondered why a battered wife returns to her alcoholic spouse only to be battered again?

Have you ever agonized over how to create change in an alcoholic family?

Drews makes a promise: "After making the changes outlined in this book, you will find yourself living a sane and serene life whether your alcoholic is still drinking or not." It is obvious that this statement is addressed to the alcoholic's spouse, but the book should be considered required reading for everyone who works with alcoholics and their families, for AA and Alanon members, for interested persons, and especially for all members of alcoholic families.

The concepts defined in *Getting Them Sober* are "difficult yet simple." They are reminders that changing is a difficult process, regardless of the simplicity of the solutions.

Drews combines incisive detail about the alcoholic personality with practical, realistic behaviors for those significant persons affected by another's alcoholism. This information will help them to adapt, and may impact the alcoholic to begin his/her own walk to wellness. Alcoholism and its effects on the family are analyzed with sensitivity and dignity, and practical and useful working guidelines are provided for those who desperately need them. Readers will find direct advice regarding many specific situations, most of which are likely to be familiar. This book does not leave the reader asking how or why.

Although the concepts found here are specifically related to alcoholics, they are applicable to other types of drug abuse problems as well.

This book can make a significant contribution in treatment centers, AA groups, courtrooms, and homes. The author's promise is well-fulfilled. — *R.M.*

Aging in America: Perspectives on Health

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Medicare and Medicaid programs spend $61 billion annually for health care for the elderly and handicapped, yet beneficiaries incur additional expense of nearly 20 per cent of their income. This conference offers the opportunity for participants to explore the critical issues relating to the provision of adequate care with maximum efficiency.

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Registration information is available from the Western Gerontological Society, 833 Market Street, Suite 516, San Francisco, CA 94103; 415-543-2617.

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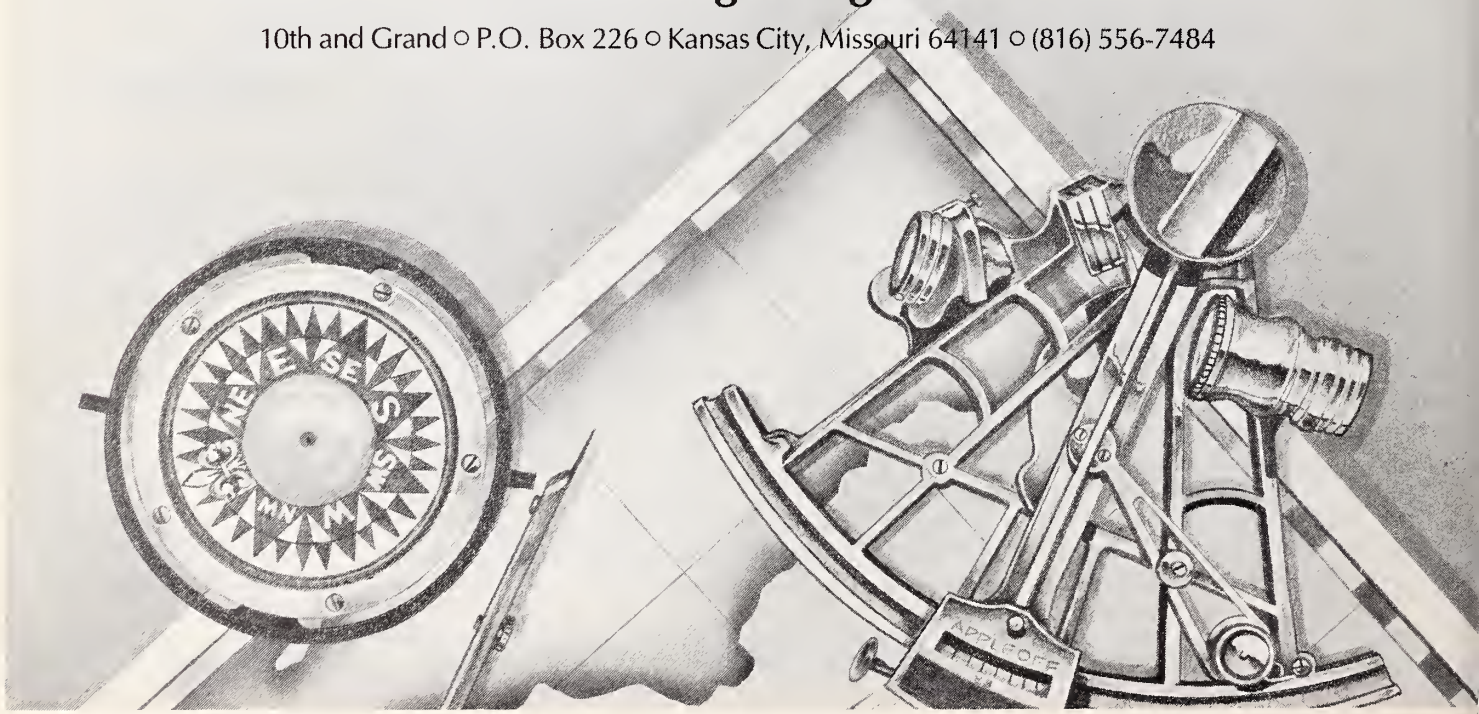
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AUXILIARY NEWS

An Open Letter to Kansas Physicians

The American Medical Association has asked the AMA Auxiliary to join in a nationwide program to combat child abuse and neglect. The focus of the Shape Up for Life campaign is the Auxiliary's child abuse initiative.

KMS Auxiliary members are encouraged to work with the Kansas Committee for Prevention of Child Abuse (KCPCA) during the 1983-84 campaign. There are 25 coalitions in Kansas involved in child abuse prevention efforts. If there is not one in your community, help is available from KCPCA to implement prevention programs.

The Kansas Committee for Prevention of Child Abuse and Kansas Action for Children are sponsors for the seventh Governor's Conference for Prevention of Child Abuse and Neglect at the Canterbury Inn, Wichita, October 13-14. The theme of the Conference is "Strengthening Families." Dr. James Garbarino will speak on "Protecting Children from Child Abuse and Neglect — Developing and Maintaining Effective Support Systems for Families."

KCPCA, Bubbylonian Productions, and the Johnson County Mental Health Center have jointly produced a film — "Bubbylonian Encounter" — dedicated to the prevention of sexual child abuse. It is designed especially for elementary school children, who constitute one-half of all sexual assault victims, and is now being used in 42 states.

The film begins with the arrival of Bub from the planet Bubbylonia, whose inhabitants are deprived of the sense of touch. "I can see velvet, spring rains, summer breezes, soft hands, and fuzzy kittens," Bub remarks, "but I can't feel them." With the aid of two new earth friends, Archie and Betty, Bub begins to discover the wonders and perils of touching.

"Lord of the Locks," produced in 1982 by the Kansas Committee for Prevention of Child Abuse, was created to help latchkey children and their parents to improve self-care situations. As the Bernardis struggle to resolve the problems faced in caring for themselves, they offer role models for real families dealing with the same issues.

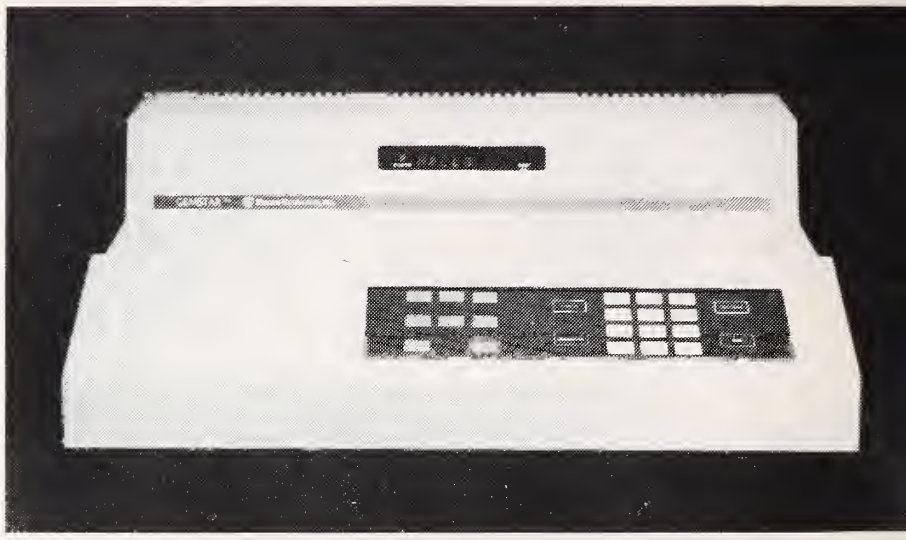
The trials of the beleaguered family in the film are experienced by many families in which parents are away at work or other activities. There is increasing evidence of physical and emotional harm encountered by children in latchkey situations. The risks for children left in self-care are undeniable. Unsupervised children are more likely to be victims of accidents and sexual molestation. Emotionally, latchkey children often feel socially isolated and fearful of being alone.

"Lord of the Locks" and "Bubbylonian Encounter" are films available for rent or purchase from the Kansas Committee for Prevention of Child Abuse. For additional information, contact January Scott, Executive Director, KCPCA, 214 W. 6th Street, Suite 301, Topeka 66603, 913-354-7738; or Wanda Macy, Rt. 3, Box 381, Salina 67401, 913-823-8966.

Wanda Macy, Co-chairman,
KMS Auxiliary Committee for
Prevention of Child Abuse; and
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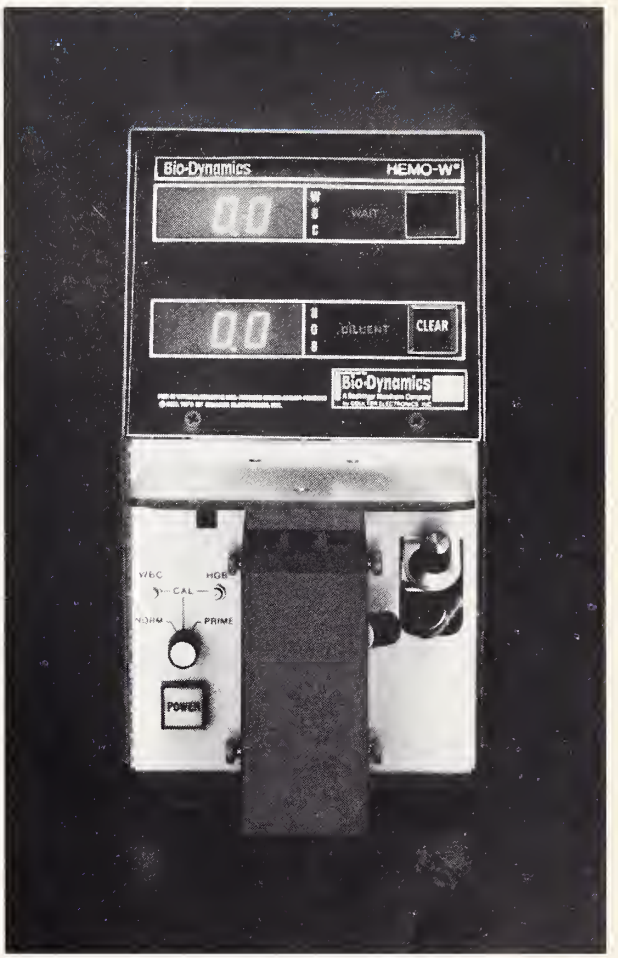
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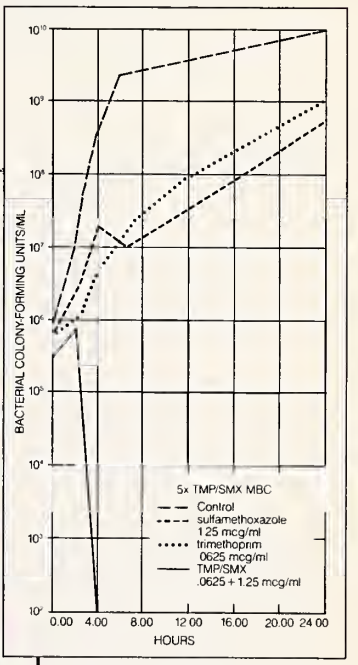
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ampicillin	72% 345,209	5% 65,466	85% 67,338	16% 4381	70% 4402	10% 1775	9% 7759	11% 12,091
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†Analogous to cephalothin, the primary antibiotic disc used in testing.
Source: The Bacteriologic Report, BAC-DATA Medical Information Systems, Inc., Winter Series, 1981-82.
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See next page for references and a summary of product information.

References: 1. Data on file, Hoffmann-La Roche Inc., Nutley, NJ. 2. Kramer MJ, Mauriz YR, Robertson TL, Timmes MD: Morphological studies on the effect of subinhibitory and inhibitory doses of sulfamethoxazole-trimethoprim combination on *Escherichia coli*. Presented at the 12th International Congress of Chemotherapy, Florence, Italy, Jul 19-24, 1981. 3. Spicehandler J *et al: Rev Infect Dis* 4:562-565, Mar-Apr 1982. 4. Stamey TA: *Pathogenesis and Treatment of Urinary Tract Infections*. Baltimore, Williams & Wilkins, 1980, p. 13. 5. Ronald AR: *Clin Ther* 3:176-189, Mar 1980. 6. Cooper J, Brumfitt W, Hamilton-Miller JMT: *J Antimicrob Chemother* 6:231-239, 1980. 7. Gower PE, Tasker PRW: *Br Med J* 1:684-686, Mar 20, 1976. 8. Cosgrove MD, Morrow JW: *J Urol* 111:670-672, May 1974. 9. Iravani A *et al: Antimicrob Agents Chemother* 19:598-604, Apr 1981. 10. Schaeffer AJ, Flynn S, Jones J: *J Urol* 125:825-827, Jun 1981. 11. Rous SN: *J Urol* 125:228-229, Feb 1981. 12. BAC-DATA Medical Information Systems, Inc., Bacteriologic Reports, Winter Series, 1976-82.

Bactrim™ DS

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:
Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.
For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.
For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.
For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.
Also for the treatment of documented *Pneumocystis carinii* pneumonitis.
Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.
Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, hepatocellular necrosis, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.
Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients. *Pregnancy:* Teratogenic Effects: Pregnancy Category *C*. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.
Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, hepatocellular necrosis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.
Dosage: *Not recommended for infants less than two months of age.*
URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:
Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.
Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.
For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.
ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:
Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.
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Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.
Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 20. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. *Pediatric Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz (1 pint). *Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per tea spoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).

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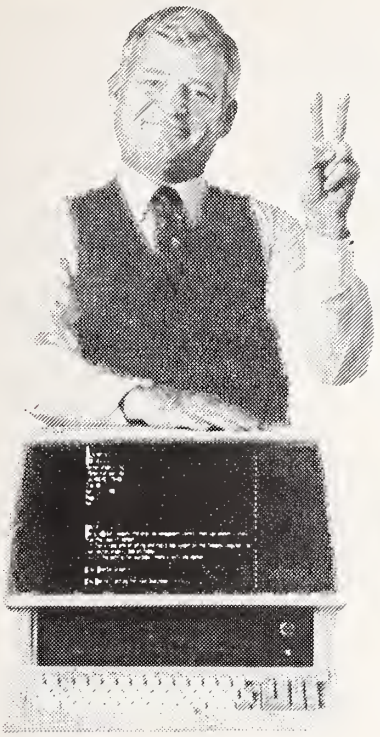


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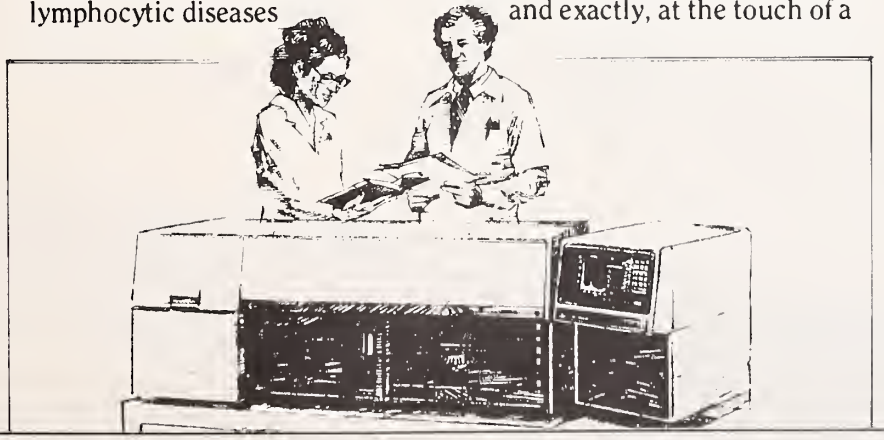
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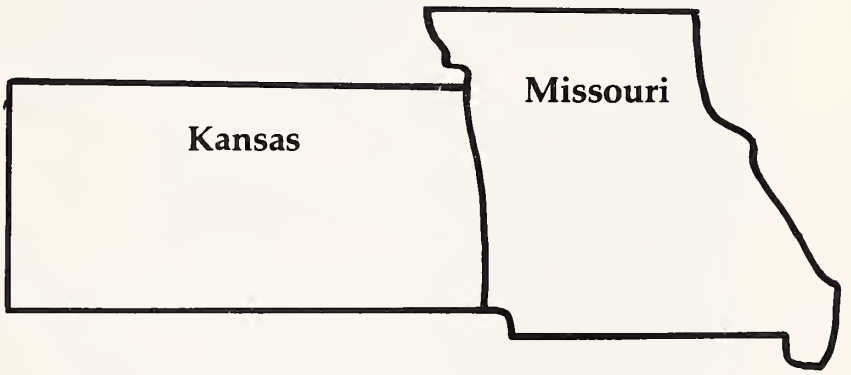
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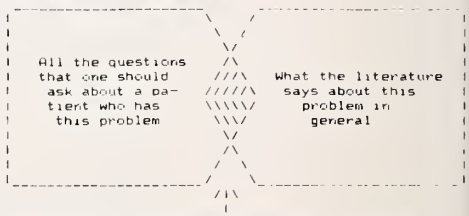
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Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile.* Other causes of colitis should be ruled out.

Precautions: *General Precautions*—If an allergic reaction to Ceclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers—Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor® (cefaclor, Lilly) is administered to a nursing woman.

Usage in Children—Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below:

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations of SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

[061782R]

* Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae.*[8]

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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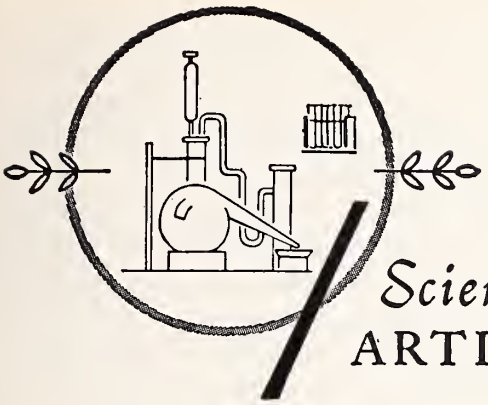
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Esophageal Cancer

Audit of Cases at St. Francis Regional Medical Center, Wichita

RUTH S. TAYLOR, M.D.,* *Wichita*

CARCINOMA of the esophagus carries a poor prognosis in the United States because it is seldom diagnosed at a stage when it is amenable to treatment. Medical records were studied for patients diagnosed at St. Francis Hospital, Wichita, from January 1, 1970, through December 31, 1979. The study was limited to cases of squamous carcinoma because of the difficulty in accurately determining whether cases of adenocarcinoma were arising in the esophagus or were extensions of primary lesions in the cardia of the stomach.

There were 63 patients with squamous carcinoma, ranging in age from 42 to 88 years (mean age 64.5 years). Race and sex distribution is shown in *Table I*. The disease occurred more frequently in males in both blacks and whites, with an overall male:female ratio of 3.4:1.0 (2.8:1.0 for whites; 5:1 for blacks).

* Cytopathologist, Department of Laboratories, St. Francis Regional Medical Center, Wichita, and Assistant Clinical Professor, Pathology, The University of Kansas School of Medicine-Wichita.

TABLE I
SQUAMOUS CARCINOMA OF ESOPHAGUS
Distribution of 63 Cases According to Race and Sex

	Totals	*Male*	*Female*
White	57	42	15 (2.8:1)
Black	6	5	1 (5:1)
	63	47	16 (3.4:1)

A ten-year review of 63 cases of squamous carcinoma of the esophagus revealed that only six patients survived more than five years. Based on experience in China, it is suggested that cytologic study and routine esophagograms among high risk persons might permit earlier diagnosis and treatment and improve the prognosis.

History and Symptoms

All patients in our study presented with symptoms of the disease. As might be expected, the most consistent symptoms were dysphagia and dyspepsia, which were recorded in 61 of the 63 patients. Forty-three (68%) had experienced recent weight loss. No other consistent symptom or finding was noted. One patient gave a history of previous ingestion of a caustic substance. Two patients had had previous head and neck malignancies (tongue and pharynx) treated with radiation therapy.

Smoking history was missing in 22 per cent, and 17 per cent lacked any notation concerning alcohol consumption. Of those whose histories were recorded, 90 per cent of the patients were smokers and 71 per cent had histories of prolonged drinking. More than one-half of these had combined smoking and drinking histories.

TABLE II
SQUAMOUS CARCINOMA OF ESOPHAGUS
Methods of Diagnosis

Diagnosis	*Tissue Biopsy Cases*	*Brush Cytology Cases*	*Esophagogram Cases*
Positive	53 (85%)	25 (89%)	42 (75%)
Negative	9 (15%)	3 (11%)	6 (11%)
Extrinsic Mass	N/A	N/A	8 (14%)
Total cases diagnosed	62 (100%)	28 (100%)	56 (100%)
Results not on chart	1	1	2
No specimen	0	24	5
Total cases	63	63	63

Diagnosis

The most consistent diagnostic tool was esophageal biopsy, which was performed on all 63 patients. Esophagograms were obtained on 58 patients. Esophageal brush specimens were obtained from only 29 patients. *Table II* compares the accuracy of diagnoses by these three modalities: cytology 89 per cent; tissue biopsy, 85 per cent; and esophagogram, 75 per cent. The presence of an extrinsic mass was diagnosed by esophagogram in eight other cases (14%). It is of interest to note that of the nine negative biopsies, four were diagnosed as positive by cytologic study, two were negative, and a cytologic specimen was not obtained from three patients.

Of the three negative cytology diagnoses, one tissue biopsy was positive. For two patients, cytology and biopsy specimens were both negative. The location of the esophageal lesions is shown in *Table III*. Almost one-half of the lesions (47.6%) occurred in the middle third, with roughly 25 per cent each in the lower and upper thirds of the esophagus.

Therapy

Methods of treatment are given in *Table IV*. Radiation therapy alone was the most frequent mode of treatment, comprising 31 of 57 (54%) cases. Surgical procedures were the primary treatment for 11 patients. In none of these was the lesion located in the upper third of the esophagus. Twenty-three additional patients underwent secondary palliative operations, essentially to relieve obstruction. In five patients, a permanent esophageal tube was inserted.

Combined modality treatment composed of radiation therapy and surgery or chemotherapy was given to 15 patients. The choice of therapy probably depends in many cases on the location of the lesion. Lesions in the upper third of the esophagus do not lend themselves to surgical procedures.

TABLE III
SQUAMOUS CARCINOMA OF ESOPHAGUS
Location of Lesion

Upper 1/3	14 (22.2%)
Middle 1/3	30 (47.6%)
Lower 1/3	19 (30.2%)

Survival

A comparison of the survival rates for this study to those of a study of 1,002 cases of esophageal cancer in Denmark for the years 1954-1969 is shown in *Figure 1*.[1] All types of esophageal cancer were analyzed in that study.

In our study, more than 54 per cent of the 59 cases whose survival is known were dead within six months, and 73 per cent by the end of one year. However, of the nine patients who survived for 24 months, six were still alive after five years. In the Denmark study, 50 per cent of the patients were dead by 5.2 months, and the five-year survival rate was only 2.3 per cent.

An attempt was made in our study to determine whether the location of the lesion or the method of therapy significantly affected survival. No conclusions could be drawn from these data. One reason, probably, is the small number of cases in each category.

TABLE IV
SQUAMOUS CARCINOMA OF ESOPHAGUS
Mode of Therapy by Site of Lesion

Therapy	*Total*		*Esophagus* *Upper*	*Middle*	*Lower*
Surgery only	11	(19%)	0	8	3
Radiation only	31	(55%)	9	15	7
Radiation & chemotherapy	4		0	2	2
Surgery & radiation	5	(26%)	0	2	3
Surgery, radiation & chemotherapy	6		1	1	4
	57	(100%)	10	28	19
Unknown	6		4	2	—
Total cases	63		14	30	19

Secondary Palliative Surgery: 23
Insertion of Permanent Esophageal Tube: 5

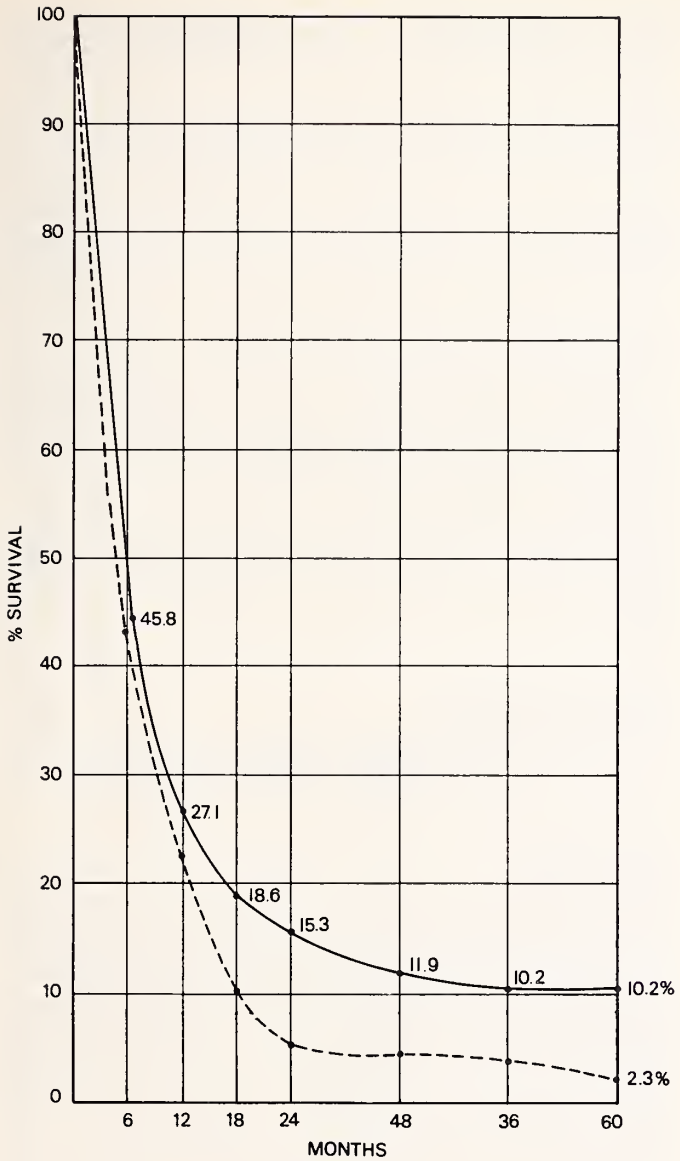


Figure 1

Figure 1. Survival rates, carcinoma of the esophagus. ——— St. Francis Hospital, squamous carcinoma, 1970-1979, 59 cases. --- Denmark, all carcinoma, 1954-1969, 25 years, 1002 cases.[1]

Table V lists six patients who survived five years or longer by sex, race, age of diagnosis, location and grade of lesion, and therapy. No consistent attribute contributed to their survival. Four of the six who survived for five years were women. For four patients, the primary therapy was surgery. Localization of lesion for four patients was in the middle third, with one each in the upper and lower third of the esophagus. One-half of the cases were histologic Grade II. At least three were without evidence of disease at their last followup. Interestingly, the table shows that of these six patients, one patient has survived for more than ten years, and another more than eight years. Both are female, and in both patients the lesion was located in the middle third of the esophagus and had been treated by operation only. Although the stage of the lesion could not be accurately ascertained, histologically both were Grade II lesions.

Discussion

In the United States, carcinoma of the esophagus is predominantly a disease of males. The incidence has risen by 15 per cent during the past 30 years. The 1978 mortality rates were 5.4/100,000 for men and 1.5/100,000 for women. The overall male:female ration was 2.4:1.1.[2] The ratio was 3.4:1.0 in our study. In some countries the incidence is much higher, *e.g.* China, Japan, Finland, and Africa. However, in one area of Asia the disease occurs much more frequently in females than in males.[3] Environmental factors undoubtedly play a role in these areas.

Smoking (especially cigarettes) and alcohol consumption are considered etiologic agents for carcinoma of the esophagus. Also, cigarette smoking in combination with alcohol increases the risk. Poor nutritional state is probably an added factor.[4] Histories about smoking and alcohol consumption were

TABLE V
SQUAMOUS CARCINOMA OF ESOPHAGUS
Patients Who Survived 60 Months or Longer

Race, Age, Sex	*Location of Lesion*	*Histologic Grade*	*Treatment*	*Length of Survival*	*Comments*
B65F	Middle	II	Surgery	129 mos.	Alive 5/82; no evidence of cancer
W64M	Lower	III	Surgery/radiation 5 years later	67 mos.	Died
W60M	Upper	III	Radiation	67 mos.	Alive 4/82; no evidence of cancer
W59F	Middle	II	Surgery	88 mos.	Alive 5/82; no evidence of cancer
W57F	Middle	Biopsied elsewhere	Radiation	61 mos.	Died
W50F	Junction middle & distal	II	Surgery	60 mos.	Alive 8/82

missing for approximately 20 per cent of our patients. Of the histories present, an overwhelming majority were positive for one or the other, and more than one-half were positive for both smoking and drinking.

Achalasia is also associated with an increased incidence of esophageal cancer.[5, 6] We were unable to abstract this information from the charts in this series of patients. Carcinoma of the esophagus has an increased incidence among persons with a history of ingesting caustic substances, *e.g.* lye,[6, 7] and also in those who have had previous neck and head cancer.[6] One person in our study had previously ingested caustic material and two had previously had head and neck malignancies. Other entities which have at least a suspected association with esophageal carcinoma are hiatal hernia, pulsion diverticulum, tylosis, Plummer-Vinson syndrome, celiac disease, and Barrett's esophagus.[6]

Carcinoma of the esophagus is a dismal disease. The lesion is seldom diagnosed early, since patients tend to ignore mild symptoms of dysphagia and dyspepsia for many months before seeking relief.[8] In our series of patients, no case of carcinoma-in-situ was found. According to *ACS Facts and Figures,* the estimated deaths in 1982 from carcinoma of the esophagus (8,300) are nearly as many as the estimated new cases (8,900),[2] giving further support to the seriousness of the disease.

Survival rates also attest to this fact. In the United States, the five-year survival rate of 4 per cent has not changed during the past 20 years.[2] The five-year survival rate was considerably higher in our study (10.2%); however, the numbers are too small and evidence is lacking to make definite claims that earlier diagnosis or therapy may have been reponsible, although it is suggestive.

According to Shu,[9] in China — which has one of the highest incidences of esophageal cancer in the world (1978 age-adjusted mortality rates of 32/100,000 for males and 16/100,000 for females) — screening for early cases is being successfully done with a high degree of accuracy using a modified Panico gastric balloon. Prior to 1979, more than 500,000 people were examined with a detection rate of more than 90 per cent in outpatient clinics and about 80 per cent in mass screening programs. Many cases of early esophageal cancer and precancerous lesions have been found. As a result, the five-year survival rate has risen from 10 per cent in the 1950s to 30 per cent in cases of advanced cancer and 90 per cent in early cancers in the 1970s.

Since the esophagus is not a major cancer site in Americans, such screening methods are not likely to become popular in the near future. However, among the high risk population groups referred to above, cytologic screening studies and routine esophagoscopies might improve the dismal prognosis of the disease by permitting earlier diagnosis and treatment.

Acknowledgements

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Neuroleptic Malignant Syndrome

Association With Thioridazine Hydrochloride in a Manic Depressive Patient

STUART W. TWEMLOW, M.D.,* *and* GLENN O. BAIR, M.D.,† *Topeka*

Neuroleptic malignant syndrome (NMS) is an underdiagnosed, potentially lethal consequence of treatment with potent neuroleptic drugs.[1] NMS has not yet been fully characterized, although authorities such as Caroff[1] and Smego[2] consider that hyperpyrexia, alterations in consciousness, muscular rigidity, and autonomic dysfunction are the primary attributes of the syndrome. NMS is associated with a wide variety of the major neuroleptic drugs, including the phenothiazines, and long acting depot phenothiazines.[2] It usually occurs after therapeutic rather than toxic doses of the drug and is apparently unrelated to the duration of therapy.[1, 2]

The confusion in the literature regarding the clinical and laboratory characteristics of the syndrome is illustrated by the differing points of emphasis authors give. Delay and Deniker[3] emphasize respiratory distress as the cardinal feature of NMS. Caroff[1] presented two cases that lacked muscular rigidity and one in which the temperature was quite low and could be ascribed to other complications. Frequently described laboratory findings are elevations of CPK, leukocytosis, and sometimes generalized electroencephalographic abnormalities suggesting diffuse metabolic encephalopathy.[2] A variety of autonomic disturbances include sialorrhea, dyskinesias, dysphagia, and diaphoresis. Often there is tachycardia, labile blood pressure, and occasionally urinary incontinence. The state of consciousness appears to vary from hyperalertness to later coma. The disease develops rather explosively during a three-day period with coma ensuing, and may last up to two weeks after cessation of the drug. Caroff[1] reports that young adult males predominate among reported cases, and that 80 per cent of patients are under age 40 years. Patients with organic brain disease are thought to be at high risk for developing NMS, but a wide range of psychiatric diagnoses have been reported, including alcoholism, schizophrenia, characterological diagnoses and, in one case, there was no history of psychiatric disorder.

Only one case similar to ours has been reported in association with thioridazine hydrochloride.[4] In this case, the patient was thought to have malignant hyperthermia as an allergic reaction to thioridazine.

This case of neuroleptic malignant syndrome in a manic depressive female patient illustrates the main clinical features of the syndrome, although association with thioridazine hydrochloride is unusual. The syndrome represents a final common pathway for a complex variety of physiological and genetic factors present during heightened susceptibility to the neuroleptic drug.

Case Report

A 70-year-white widowed female had been periodically hospitalized for manic depressive psychosis since 1940, spending approximately 28 years in institutional settings. She had received in excess of 80 electroshock treatments since 1940. Approximately three years ago, she was admitted to a psychiatric intensive nursing care home where she came under our care. Her illness is characterized by well established cycles of about two weeks of mania, including flights of ideas, grandiose fantasies, and psychomotor agitation with insomnia. Periods of depression last about six weeks and have been accompanied by at least three suicide attempts since her first admission. When depressed, she becomes angrily negativistic and withdrawn, fails to eat, and loses weight.

Her past medical history includes chronic congestive heart failure secondary to mitral incompetence first diagnosed in April 1981 and periodic labile hypertension. She also has had a hysterectomy for fibroids and has a recurrent chronic urinary tract infection.

She has had two previous psychiatrically supervised clinical trials of lithium carbonate, each of

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which was teminated when she developed a serious cardiac arrhythmia. Since that time, her psychiatric management has included careful titration of the tricyclic antidepressant imipramine hydrochloride in doses ranging from 50 to 300 mg/day and thioridazine hydrochloride in doses ranging from 100 to 300 mg/day. With this management, there was evidence that in the past 18 months, her mood swings had become less extreme.

The nursing staff noted that the patient entered a depressed cycle in April 1982. Her progressive "depression," however, was seen to be different by staff who had known her for many years. She was not her usual angry, negativistic self, but was more withdrawn and extremely fearful with periodic comments that she was about to die. Approximately two weeks later, there were frequent references in the nursing progress notes that the patient was drinking a lot of water and was quite cool and clammy at times. She was apyrexic. These episodes of clamminess were often accompanied by lethargy and failure to understand or acknowledge anything said to her and a hostile resistance when staff would attempt to aid her. As her mood became more depressed, imipramine hydrochloride was increased to 300 mg at bedtime. Her blood pressure continued to be labile, ranging from 104/70 mm Hg to 160/100 mm Hg. Her pulse rate varied from 68 to 100/min on digoxin and Inderal. On the day of admission to an acute care hospital, her blood pressure was 160/100 mm Hg; regular pulse, 96; respiration rate, 22/min; and temperature, 37.5C. The nursing staff noted that she fluctuated in alertness, appearing comatose at times, but responsive to painful stimuli. She remained passively cooperative in bed, but did not attend to her ordinary bowel and bladder hygiene. While not incontinent, she required catherization and intravenous fluids to reestablish fluid balance. At first, it was thought that she might have suffered a transient cerebral ischemic episode, since staff noticed that her head was pulling to the left and she seemed weak on the left side. In view of her complicated medical condition, other diagnoses such as pulmonary embolism and cardiac arrhythmia were also entertained. Nursing staff commented most regularly on two main clinical symptoms: (1) A plastic rigidity of the limbs that was not like the "waxy flexibility" of catatonic schizopherenia. There was a mild extrapyramidal tremor and no cogwheel rigidity of the limbs. There was an increase in dyskinetic movements of the tongue with sialorrhea, which had been present for some two years previously. (2) A profuse diaphoresis was present similar to that seen in acute hemorrhagic shock. This diaphoresis was the earliest sign of NMS, and the first to resolve, thus giving a potentially useful barometer of the syndrome.

The initial laboratory investigations showed no marked abnormalities. Creatinine phosphokinase was entirely normal. There was mild elevation of alkaline phosphatase of 482 IU/L. In addition, white count was 8,500/cu mm with a normal differential and normal red cell morphology. Her thyroid profile, including T_4 and T_3, was normal. Her digoxin level was in the therapeutic range. Serology was nonreactive; urinalysis was normal. She had a sedimentation rate of 24 mm/hr. Her EKG showed left ventricular hypertrophy with no significant change from previous EKGs. Chest x-ray showed mild cardiac enlargement and no acute cardiac or pulmonary pathology, and her EEG was entirely normal. In the course of her 11-day hospitalization, her temperature reached its highest point on the second day after admission at 38.1C and fluctuated between that range and approximately 37.2C. At this point, she was semi-comatose, although her blood pressure remained stable, ranging between 120-140 to 90-100 mm Hg. Her respiration rate was moderately elevated at 20 breaths per minute. Her pulse varied between 64 and 100 beats per minute.

NMS had not been considered in the initial differential diagnosis, since her psychiatric state gave no cause for alarm. The dose of thioridazine on admission was 50 mg two times each day, one of the lower levels that had been recently used. Once NMS was suspected, the thioridazine was immediately stopped; all other medications were maintained. Within 24 hours, her clinical condition began to improve dramatically, as signalled by the immediate cessation of diaphoresis. She gradually returned to a normal sleeping pattern, regained her appetite, and became talkative, cooperative, and ambulatory. Her temperature returned to her normal low level of 35.0 to 35.6C.

Since her discharge six months ago, the patient has not had a return of symptoms. Interestingly enough, approximately one month after discharge, she developed an acute flare-up of her chronic urinary tract infection, with profuse sweating quite clinically different from the peculiar drenching diaphoresis noted at the time of the NMS.

Discussion

The patient is an older woman, ingesting a multiplicity of drugs, and with a variety of chronic medical illnesses thus creating considerable diagnostic difficulty. The problem is further complicated in older people, because symptoms such as hyper-

pyrexia, considered as a cardinal sign of NMS, might not be as high as in younger patients, because older people do not always show marked temperature elevations in response to physical illness. The diagnostic problem is made even more difficult because the symptoms and signs of NMS are not related to the dosage or onset of use of the neuroleptic drug. The time course of the illness in this case followed a "classical" course.[1, 2] Rehydration with intravenous fluids and cessation of the thioridazine hydrochloride resulted in a complete recovery in five days, the time period usually reported. Rehydration alone did not alter the course of the illness. Both literature and clinical material indicate that what predisposes the patient to this syndrome is a physiological change in susceptibility to the neuroleptic drug. Otherwise, it would be expected that the syndrome would relate closely to onset of use of the drug as it is with most extrapyramidal drug reactions.

Itoh[5] reviewed fourteen cases of NMS. He noted that all cases suffered from physical exhaustion and dehydration prior to the onset of the syndrome. The most prominent clinical symptom was profuse diaphoresis, very similar to that seen in cardiovasuclar collapse. In all cases reported, no such cardiovascular collapse exists, although blood pressures and pulse often fluctuate.

The problem of differentiation from acute lethal catatonia remains the most difficult. Meltzer[6] discusses this diagnosis and points out that at times it can be virtually impossible to distinguish it clinically. Characteristic catatonic "waxy flexibility" is different from the rigidity in this syndrome which is much more like Parkinson's disease. The differentiation from anesthetic-induced malignant hyperthermia is also important. This condition is rare, always associated with anesthetic use, and has a much more prominent fever. The CPK elevation frequently reported in NMS is probably a function of muscle cell damage.

Manic depessive psychosis is generally agreed to be a partially genetic disorder and is seen by some authorities as a disorder of central amine modulation.[7] Since it is known that the neuroleptic drugs are dopamine receptor blockers, then manic depressive patients who have poor modulation of the critical balance of amines in the central nervous system might be particularly susceptible to neuroleptics. Others[4] have suggested an allergic mechanism. Allergy has some plausibility if one considers as a model a delayed hypersensitivity reaction with increasing sensitivity to repeated drug exposures.

The syndrome can be conceptualized as a *final common pathway* with a variety of etiologic mechanisms, each involving a critical balance between the neuroleptic drug and the physiologic state of the patient. This state is affected by: disturbances in amine balance in the central nervous system in mood disorders; fatigue and dehydration affecting ion transportation; organic brain syndrome affecting cortical sensitivity; delayed hypersensitivity reaction; and other drugs affecting amine modulation — for example, the tricyclic antidepressants — and drugs, including neuroleptics, that can affect the homeostatic temperature regulating function of the anterior hypothalamus.

A recent paper by McCarron[8] reports the successful use of amantadine in the treatment of NMS syndrome. This drug is thought to increase synaptic dopamine availability by antagonizing the dopamine blocking effect of neuroleptic drugs, thus providing a potential model for understanding the mechanisms of the illness. There is a similarity between NMS and the well recognized lithium neuroleptic encephalopathy.[9] In lithium neuroleptic encephalopathy, patients develop an EEG-demonstrable encephalopathy with combined use of lithium and major neuroleptics, most commonly haloperidol. While not developing high temperatures, these patients usually have some of the other signs of the neuroleptic malignant syndrome.

In general, NMS appears to be a *final common pathway* for a complex variety of preexisting multidetermined conditions. The syndrome should always have a prominent place in the differential diagnosis of stupor or coma of apparently undetermined etiology.

Acknowledgements

Staff and residents at Howe Psychiatric Care Center, Topeka, contributed to the understanding of this material. Joan Pagel prepared the manuscript.

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(Continued on page 533)

The Hammock Position

A Possible Aid in Prophylactic Treatment of DVT

IRA R. MORRISON, M.D.,* *Atchison*

ARCHIMEDES discovered that the volume of irregular shaped objects could be determined by measuring the amount of water they displaced. A similar technique, which allows measuring the volume of a leg, was first used in the Atchison Hospital, then at Mount St. Scholastica Convent in 1977. It was attempted in an effort to find a simpler method to detect deep venous thrombosis (DVT) and to gain experimental experience by measuring the volume of each leg of various individuals.

A leg afflicted with DVT usually has an occult increase in volume which is difficult to assess visually. A volume change of 0-200 ml/day in each leg is not considered abnormal nor unusual, but that much change in only one leg usually is. A bilateral increase in leg volume is apt to subside during a night's rest, whereas a unilateral leg swelling is less likely to subside.

If a Plexiglas container of appropriate size and design (*Figures 1, 2,* and *3*) is filled with water to a siphoned depth of 15 inches, and both container and water are weighed together, this weight may become a standard measurement in determining leg weight per volume. If while standing, sitting or reclining, a leg is placed into an empty or partially filled container which is then filled with lukewarm water up to a siphoned depth of 15 inches, and the leg is then removed, the weight of the container and the remaining water subtracted from the weight of the container filled to a 15-inch siphoned-level depth will indicate the leg volume. The unconscious patient may need to have the leg inserted into the container, the bed lowered, and the water added.

A heart-low or hammock position may be useful in the treatment of various circulatory problems. One such affliction — DVT — may be diagnosed with a simple water displacement device to determine volume of the leg.

The dominant leg (usually the right in right-handed subjects) is commonly about 0-100 ml larger than its fellow. The volume of a leg may vary greatly during a 1-to-12-hour period in a normal adult. These changes may not be important, but an increase in volume of only one leg may be. The drainage carried out by a siphon should require less than one minute; the volume evaluation of both legs with the help of a technician usually requires less than five minutes. Performance of the task unaided takes longer.

Any scale that weighs 0-15 kg or 0-33 pounds in fractions will usually suffice, or the complete siphon overflow collected as the leg is slowly immersed into the tank may be collected and weighed; it may also be measured in volumetric vessels.

The primary piece of equipment is a 6-inch inside diameter clear vinyl Plexiglas tubing 17 inches long; a larger leg may require a 7-inch inside diameter tubing. The tubing is modified at the base to make room for the instep, foot, and toes. Nurses have dubbed the container "The Invisible Boot." It holds more than a 15¼-inch depth of water before it activates the siphon that drains the water down to a depth of 15 inches. A common wetting agent (hexachlor-

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Address reprint requests to Dr. Morrison, 825 No. 10th Street, Atchison KS 66002.

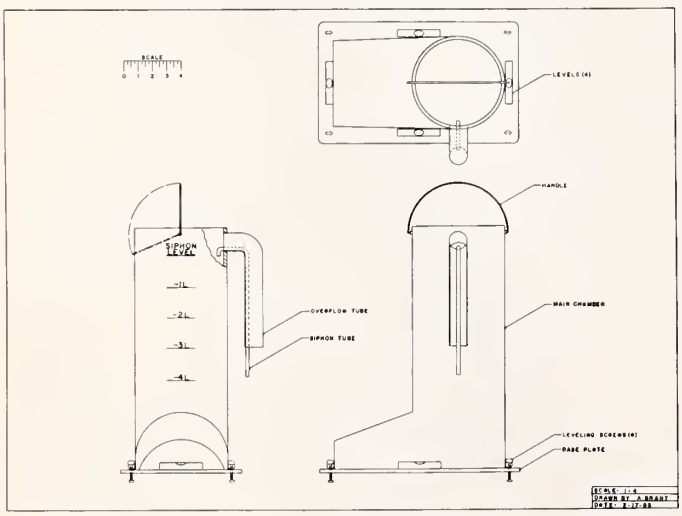


Figure 1. A sketch showing the construction of the leg container.

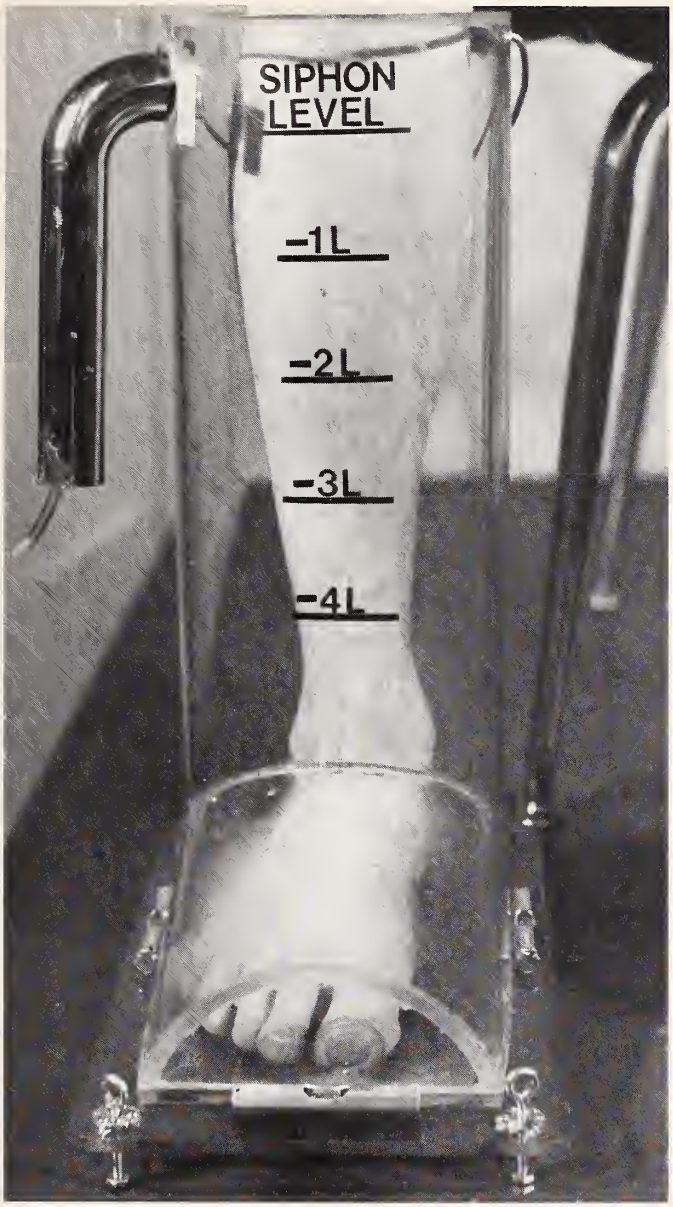


Figure 2. A photo of a leg in an empty invisible boot. No water was added due to the photographic distortion it produces.

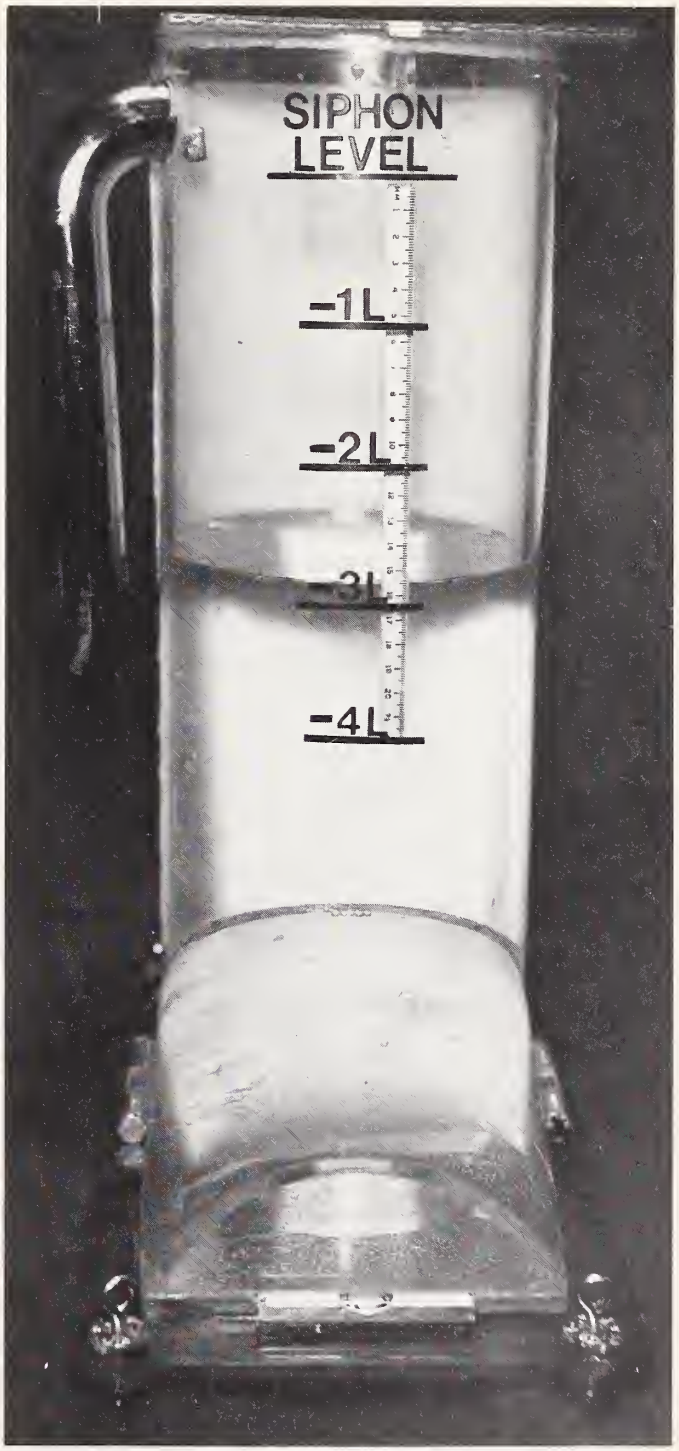


Figure 3. A photo showing the "dip stick" to facilitate estimation of the amount of displaced water. It measures the depth of water in millimeters on one side and in inches on the other.

ophene or a similar liquid surgical soap), one-half ounce per container, is useful in diminishing the .0082 pounds/inch surface tension of water. The water may be at any comfortable temperature. The plastic tank should be placed on any level floor area. This position may be gained by adjusting the corner leveling bolts until all four spirit levels read out as level. A rough estimate of the fall in the water level (after the leg is removed) is 1 mm equals 18 cc, or 1/16 inch equals 28.9 cc. A vertical ruler, supported by the brim of the cylinder, dips into the water. The measurement from the siphon level down to the water level will indicate the leg volume. The ruler reads in millimeters on one side and inches on the other.

The prophylactic treatment for deep venous thrombosis remains unsettled. This affliction is fairly common to humans whose feet and legs are somewhat distant from the blood pump, and whose hydrostatic pressure in the lower deep veins of the legs falls from about 90 mmHg while standing erect

down to zero when in a supine position.

A gradual change to a sort of supine jack-knife position — heart-low or hammock position — has been suggested for the treatment of circulatory or respiratory failure. This position aids the general venous blood return and pools blood in the middle of the body near where the circuit begins. This may eventually be demonstrated to be our healthiest normal sleeping position. In this position, many large veins are collapsed. Both the blood pressure and the work of the heart are reduced.

Concerning DVT, most physicians probably believe, as *Cecil's Textbook of Medicine* states, that: "At present, prophylaxis with minidose heparin is the preferred method for patients over 40 years of age undergoing surgery."[2] On the contrary, Dr. Owen Wangensteen, who devised the Wangensteen suction apparatus, used only two wooden blocks to elevate the foot of the bed of postoperative patients into a Trendelenburg position in an effort to diminsh the frequency of DVT. His advice has gone unheeded by our American physicians and surgeons.

A book of 254 pages edited by 25 authorities entitled *Prophylactic Therapy of Deep Venous Thrombosis and Pulmonary Embolism,* DHEW Publication No. (NIH) 76-866, describes recent experimental studies. This book states that 50,000 people die yearly in the United States, but there is no mention of Wangensteen's simple prophylactic therapy.

Patients do not adjust well to a head-low position, but enjoy a hammock position. If Wangensteen had positioned his patients in a hammock position with both feet and head above the level of the heart, it is probable that his prophylactic treatment for DVT would have been accepted and followed. And many lives may have been saved.

Acknowledgements

Stanley Friesen, M.D., Professor of Surgery, UKSM-KC, who was a resident on Dr. Wangensteen's service; Sister Elaine Cranford, O.S.B., M.S., Mount Scholastica Convent; and Father Benjamin Tremmel, O.S.B., Ph.D., Instructor in Biology, Benedictine College all made valuable contributions to research and development of this material.

The Hays Sheet Metal Shop, Atchison, participated in design and development of the "Invisible Boot" for measuring leg volume.

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Drug INFORMATION

JOYCE GENERALI, R.Ph., *Editor*

Dioxin

Question: With the increased awareness and concern of the public for dioxin-containing waste sites, could you give us recent study information about its toxicities and long-term effects?

Answer: Dioxin (TCDD, 2,3,7,8-tetrachlorodibenzo-p-dioxin) is a contaminant produced during the manufacture of phenoxy herbicides, 2,4,5-T (2,4,5-trichlorophenoxyacetic acid), 2,4-D (2-4-dichlorophenoxyacetic acid), and the antibacterial agent hexachlorophene.[1, 2] In fact, there are 75 chlorodibenzodioxins including 22 possible tetrachlorodibenzodioxins, all differing in toxicity.[2] Dioxin has become an important environmental pollutant because of widespread use of 2,4,5-T in agriculture, Agent Orange in Vietnam, and most recently, waste sites in Times Beach and Imperial, Missouri.

Human toxicity has been documented in only a few cases out of many exposures. Chloracne is the hallmark of acute exposure. Carcinogenicity from long-term residence in body tissue has not been reported in humans. TCDD poisoning is associated with chloracne beginning five to eight weeks post-exposure.[3] Chloracne is a follicular hyperkeratosis (comedones) with or without cysts and pustules preceded by an increased oiliness of the skin. This fact suggests an effect on sebaceous gland secretion rather than or in addition to an effect on the duct itself.[3, 4] Almost every follicle may be involved, making it more disfiguring than adolescent acne. This condition usually subsides after 14 to 20 months.

In addition to dermal effects, victims experience colicky abdominal pain, flatulence, loss of body weight, oppressive headache, fatigue, loss of vigor, irritability, and hirsutism on shoulders, upper back, chest, eyebrows, and temporal region. Long-term effects, except for persistant chloracne, have not been reported.[5, 6] Although the mechanism is unknown, an elevation of serum chloresterol and Type 2A lipoproteinemia are frequently observed.[3, 7]

The most recent non-occupational exposure studied occurred in Seveso, Italy, on July 10, 1976.[4, 8, 9] More than 2,000 people were exposed to 0.45-6.8 kg of TCDD when a 2,4,5-T plant reactor overheated and discharged the chemical into the atmosphere. Definite chloracne developed in six weeks in 134 people, mainly children between five and six years old. An epidemiological study involving 625 women who were pregnant at the time showed no unusual incidence of pre- or postnatal mortality or morbidity.[10]

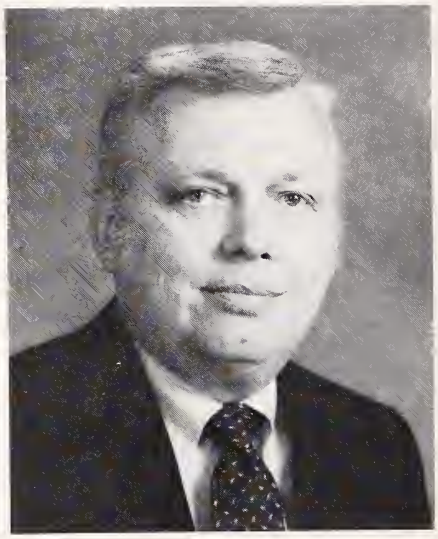
The teratogenic effect in animals of TCDD is less than with other compounds such as thalidomide and vitamin A. In animals, the teratogenic response associated with TCDD is cleft palate. TCDD tends to cause death of the embryo or fetus rather than a wide range of abnormalities.[2] TCDD accumulates in non-human primates in tissues having a high lipid content, particularly skin, muscle, and fat.[6] At present, there is no conclusive evidence that TCDD is mutagenic, teratogenic, or carcinogenic in humans.[6, 11]

TCDD is highly lipid soluble, and its major route of excretion is in feces. Dioxin in human milk sampled in areas where 2,4,5-T is used as a herbicide was undetectable at 1 ng/ml.[1, 12]

Dioxin is virtually insoluble in water and therefore almost immobile in soil. Most TCDD from surface application probably photodegrades during the first 24 hours. This has been suggested as a

(Continued on page 543)

The President's Message



October begins a new era in medical economics. The DRG (Diagnostic Related Group) system becomes operational in Medicare hospitalization reimbursement. Blue Cross and Blue Shield of Kansas will implement a similar method on January 1, 1984, for participating hospitals.

Regulations and guidelines related to all of these changes did not arrive until September. Because of this short span of time, both hospitals and physicians are likely to experience much confusion, concern, and fear. The new system itself is neither good nor bad. The determining factor will be how the system is put into place and administered.

Medical staffs must become very active in the evaluation of these programs. The study of utilization reviews, practice patterns, and cooperative ventures with hospitals and medical staff personnel must be pursued. Credentials committees, executive committees, and other ad hoc staff committees will need to be diligent in their approach to assure a fair and equitable situation. There are areas where financial savings can be achieved, but we must never let quality care be sacrificed to economics.

We physicians have a great challenge ahead of us to preserve a quality medical system. The KMS plans to keep you informed of changes and to be an appropriate aggressive organization in your behalf. We welcome your comments and ideas.

Jimmee A. Pearson M.D.

President



Editorial
COMMENT

Call Me "Mister"

Whether it is a reflex produced by sitting in front of a typewriter or a constant failure to catch up with what is going on in the world around us, we keep coming back, when confronted with the task of filling these columns, to that ubiquitous phenomenon, change. It seems to us so pervasive in all areas of our social structure that even the absence of change (brief though it may be) constitutes a form of change in itself. And now another manifestation has come to our attention.

Not long ago, the *A Piece of My Mind* department of the *JAMA* carried an offering by a medical student objecting strenuously to the custom of addressing medical students as "Doctor" during their clinical training (obviously a considerable time before they have achieved the title officially). The young man (we presume he is young and if he isn't male, she has to contend with a masculine name) declared his custom of identifying himself to patients as what he was — a student — and on only two occasions had his services been rejected on that account. Thus, in reading his comments, we couldn't help but wonder if we were witnessing still another change. Is he speaking only for himself or is there a viable feeling on the part of medical students that the title should not be prematurely applied? It seems unlikely to us that he has many adherents to his philosophy but while awaiting further developments we couldn't avoid falling into some recollections.

In our own long-ago day, we (inclusive) suffered no such compunctions about being called or calling ourselves "Doctor" in front of the patients assigned to us. Such feelings were totally unnecessary since the patients at Cook County and such places as we were permitted to impose our dubious ministrations knew very well what we were since most of them had seen many more medical students than we had patients. As if we didn't give ourselves away by our actions, our short white coats (in various states of grubbiness) with the excessively casual dangling of the stethoscope from the pocket or hooked on the neck by the ear pieces were certain hallmarks. (Now, there *is* a change. Today, so the tube indicates, one apparently wears the stethoscope draped around the neck like the skeleton of a fur piece, a custom accompanied by this strange penchant for wearing the glasses up in the hair.) In retrospect, then, being referred to as "Doctor" was not at all misleading to the patients although somewhat to us. At least it provided us with a little moral sustenance which we needed greatly.

He also refers to the fact that a nurse, misled by the title, might accept orders from a student with less than desirable results. He need have no qualms because any nurse worth her salt will have his number even before the patients. If it is his aim to maintain an attitude of propriety and self-perspective, he will do well not to worry about the title but to court both patients and nurses for their true value, his prime source of education. Of course, it could have been a little problematic, had the young man's attitude prevailed in our day, as we went to one of those schools that didn't grant the academic degree until the completion of the internship — plus whatever number of months elapsed between the end of the internship and the next convenient time to have a graduation ceremony. Had we retained the title of "Mister" all that time, we would undoubtedly have been taken for British surgeons which seems at least as reprehensible as using the undeserved "Doctor."

The student's rejection of the title is not due to a lack of appreciation for it — quite the contrary. He simply considers himself a thing apart — accepting what he is with dignity and appreciating but wanting no part of the unjustified designation. The root of his conviction is, of course, identity, that social entity that has attracted considerable attention in recent years. The business of searching for, clarifying, and exploiting identity has attained the level of an industry. The unsettled state of the Twentieth Century world has produced a massive anxiety relieved only by struggling to find ourselves with the help of

professional purveyors of identity determinations. Perhaps it is a paradox or simply a relative distortion but it seems that those individuals and institutions that were once firmest in identity are suffering the greatest misgivings and alterations, including — need we say? — medical practice.

Our friend is experiencing a fixation of identity. He has assumed an at least unusual and rigid posture which he has a perfect right to do but which makes one wonder if he is anticipating some rapturous experience at the moment he is handed his degree which will elevate him to a level matching the title. We can't help feeling he is missing something — perhaps no real impediment to his educational progress but obviously, as he notes, requiring him to take the time and trouble to inform those involved in that process of his determination. Perhaps it will enhance his feelings at the time his medical status becomes academically official but we are not prepared to agree that they will be any better for his temporal denial.

The title does have a different meaning for different people. We recall a friend from army days, a good ol' boy from some place called Nothkaylana. Our initial assignment was to a just-being-activated infantry division. We were summoned to a reception where we were to be introduced to the commanding general, a situation we viewed somewhat uneasily since brand new medical officers are as bemused by generals as generals are by brand new medical officers. As our friend was introduced, the general said, "Glad to meet you, Mister Grant." The moment passed without a call for a Second Secession but when our friend was finally coherent, he said, "Ah dint cayuh ef he dint call me 'Lootent' — ah dint wanta be one enyway, but he mote at least of recknized the fav yeahs ah spent gettin' to be a *doctuh*." We were inclined to agree but now can't recall whether the general called us anything.

If there *is* a shift in medical student identity (which we still doubt), it could well be related to the over-all change in the medical education climate which, we suspect, owes more to socioeconomic factors than professional. We look back to those between-the-wars, middle-of-the-Depression days when there was a considerable body of patients whose complete lack of financial reserves dictated their admission to medical service as charity patients. Present day sociologists are scandalized at the gross debasement of personalities the system produced in these deprived individuals but the patients, at the time, tolerated it reasonably well and, even, at times, expressed a degree of gratitude for services rendered. It seems they did not know what they were missing since they were not, like their progeny, products of a system where virtually all patients are rendered "private" because some agency or another is paying at least something toward their care. The consequence has been that the later generations could demand a level of attention free from the former indignities. It is a matter of record that the impact of this change on medical education has posed some real problems for medical educators and perhaps the rather innocent ritual of calling medical students "Doctor" is becoming one of the casualties of the new order.

But we urge the student not to spurn any expressions of professionalism, even as mild as this one, or see any corruption of person or title in the system. He even cites the question of legality by pointing out that circumstances could lead to a charge of fraud if a person is presented as "Doctor" without the official blessing. His concern is commendable but misapplied. Let us disabuse him of the notion that refusing the title will offer any protection. If he is unfortunate enough to be associated, even as a frankly defined student, in the care of a case that comes to malpractice litigation, we have no doubt the eager plantiff's attorney will include him in the net if there is any way he can be connected with the case.

So we realize that our differences in interpretation of the matter are not only of the years that separate us but shifts in mood — professional as well as public — that have come with those years. There was a time when the designation, "Doctor," immediately connoted the physician. It has, however, become commonplace. This is not to degrade those who have achieved comparable status in other disciplines (many of whom do not use it anyway) but to note the prostitution of the word by the charlatans and quacks. The misfortune is not so much their use of the term on the basis of fraudulent or inadequate or no academic preparation as the fact that a distressing portion of the public continues to be misled, thereby assuring the continued prospering of such individuals.

Whether or not our student accepts or rejects the title in his training years will not alter this but perhaps it does account for some of our sensitivity in the area. We all have our personal evaluations of the concept and defend them in our own ways. It may have come to the attention of some, for example, that in these columns we refer with reasonable consistency to "physicians" rather than "doctors" — not to downgrade the latter but to clarify the group intended. Although it has now become a matter of habit, we would hope that along the way it has served in some subtle way to remind us that the

medical purpose and dignity and title, despite assaults from various quarters, are alive and well. But they need continual sustenance and it seems to us that addressing the medical students as "Doctor," in the clinical years certainly, at a time when they are most idealistic and receptive, is really a first step in the process.

It seems particularly necessary in these days when physicians are undergoing a rigorous social reassessment. The distribution of medical functions to an increasing number of non-physicians, the increasing intrusion of outside groups into the remuneration patterns for physician services, the development of practice systems that promote limitations on the traditional relationships in the name of economy, the vulnerability of physicians to liability action, the commercialization of medical practice, all have been at once producers and products of the current medical transformation. We can assure the student that his acceptance of the title of "Doctor" during this period of his training will have little derogatory effect as compared with these forces against which he will have to defend it as long as he uses it.

Although we disagree with his position, it must certainly be taken as a measure of his sincerity and that is exemplary. If he *feels* like a medical student and not like a physician, all well and good. He is in the right place for it. But at some point, he must *feel* like a physician. Whatever his academic experience and however he manages his maturation in the process, he will eventually need a full measure of the feeling — it's not a matter of diplomas or licenses — because nothing else will sustain him so well in the medical life he is so conscientiously working toward. — *D.E.G.*

Neuroleptic Malignant Syndrome

(Continued from page 525)

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Cameo

MOONBEAMS

by G. William Nice, M.D., *Topeka*

Moonbeams are like a young mother's hands
that come softly in the night
to cover her child,
very gently lest she stir
then silently fade away into darkness.

Obituaries

Laurie J. Benz, M.D., 44, Wichita, University of Pennsylvania School of Medicine 1961, died June 19, 1983.

Stephen S. Ellis, M.D., 70, Coffeyville, Washington University School of Medicine 1936, died in November 1982.

Robert A. Haines, M.D., 66, Topeka, University of Cincinnati College of Medicine 1942, died September 20, 1983.

Cline D. Hensley, Jr., M.D., 62, Wichita, University of Kansas School of Medicine 1944, died May 7, 1983.

Herman F. Janzen, M.D., 91, Moundridge, University of Kansas School of Medicine 1935, died April 6, 1983.

Charles R. Kempthorne, M.D., 80, Manhattan, University of Wisconsin School of Medicine 1932, died May 11, 1983.

Lawrence R. Leigh, M.D., 71, Shawnee Mission, University of Kansas School of Medicine 1941, died June 12, 1983.

Robert W. Myers, M.D., 71, Newton, University of Kansas School of Medicine 1943, died March 26, 1983.

William C. Swisher, M.D., 61, Wichita, University of Kansas School of Medicine 1947, died June 17, 1983.



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1:00 - 1:05 Welcoming remarks — Dale Darnell, M.D.
1:05 - 2:20 Informed Consent — Lawrence Ferguson
Record Keeping — William Lynch, J.D.
2:20 - 3:10 "You Are Hereby Summoned To Appear" videotape, discussion
3:10 - 3:30 Break
3:30 - 4:20 From the Physician's Viewpoint — William Medlicott, M.D., Jim Gleason, M.D.
4:20 - 5:00 Recent Court Cases — Darwin Johnson, J.D.
5:00 - 5:30 Doctor/Patient Rapport — E. Ross Smith, J.D.
5:30 - 7:00 Dinner (provided)
7:00 - 8:00 Panel Discussion
Lawrence Ferguson, William Lynch, J.D., William Medlicott, M.D.
Jim Gleason, M.D., Darwin Johnson, J.D., E. Ross Smith, J.D.
8:00 - 9:00 What a Plaintiff's Attorney Looks For — Gerald Michaud, J.D.

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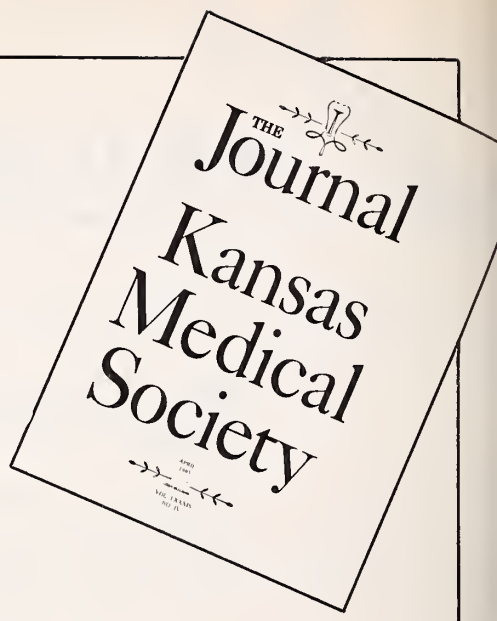
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Manuscripts must be typewritten, double spaced (all text and references), leaving wide margins. Submit the original plus one copy if possible.

Titles will be reconstituted into a three-word headline with a descriptive blurb line.

Summary: All manuscripts should include a short, factual abstract.

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Drugs should be referred to by their generic names; trade names may be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

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Bibliographic references *should be typed double spaced,* and should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation — *not* alphabetically. All references must be numbered consecutively, and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

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All material that cannot be set in type — such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) — must be identified on the back as to figure number, author's name, and an arrow indicating top. They must be referred to by number in the text, and should be numbered in order of referral using Arabic numerals. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words. Such materials should be submitted in camera-ready form, *i.e.*:

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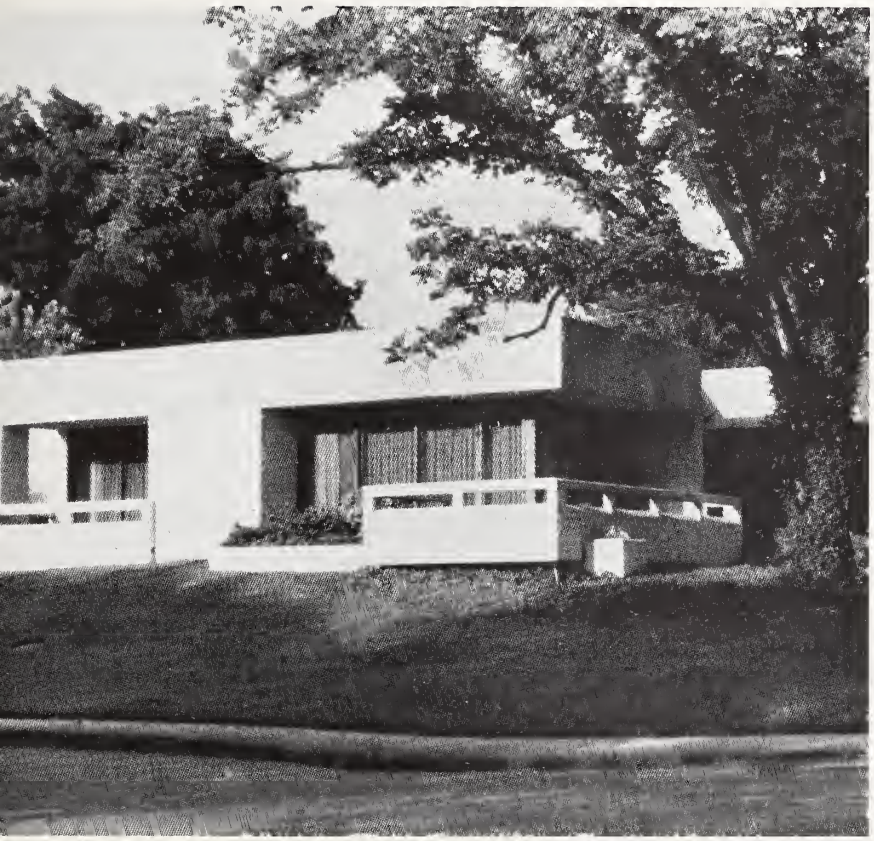
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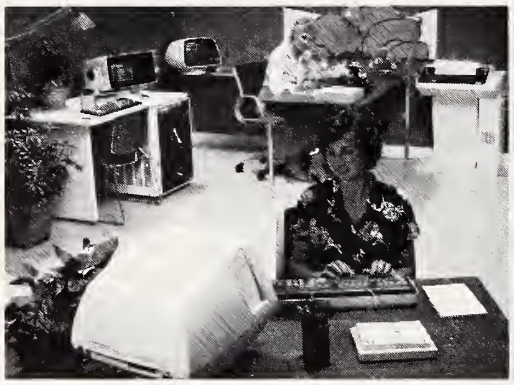
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Before prescribing, see complete prescribing information. The following is a brief summary.

DESCRIPTION: Each sustained release capsule contains 12 mg of Chlorpheniramine Maleate, USP and 75 mg of Phenylpropanolamine Hydrochloride, USP in a base to provide prolonged activity.

INDICATIONS: For the treatment of the symptoms of seasonal and perennial allergic rhinitis and vasomotor rhinitis, including nasal obstruction (congestion).

CONTRAINDICATIONS: Hypersensitivity to any of the components, concurrent MAO inhibitor therapy, severe hypertension, bronchial asthma, coronary artery disease, stenosing peptic ulcer, pyloroduodenal or bladder neck obstruction. Do not use in children under 12 years.

Do not use this drug in patients with narrow-angle glaucoma, obstructive or paralytic ileus, intestinal atony of the elderly or debilitated patient, unstable cardiovascular status in acute hemorrage, severe ulcerative colitis, toxic megacolon complicating ulcerative colitis, myasthenia gravis. Do not use in nursing mothers.

Use in treating lower respiratory tract symptoms, including asthma, is contraindicated.

WARNINGS: Caution patients about activities requiring alertness (e.g., operating vehicles or machinery). Antihistamines are more likely to cause dizziness, sedation, and hypotension in elderly patients. Patients should also be warned about the possible additive effects of alcohol and other CNS depressants.

Usage in pregnancy: Safe use in pregnancy has not been established. Use only when the potential benefits have been weighed against the possible hazards to the mother and child. Note that an inhibitory effect on lactation may occur.

PRECAUTIONS: Use with caution in patients with a history of bronchial asthma, increased intraocular pressure, hyperthyroidism, cardiovascular disease, hypertension, hiatal hernia with reflux esophagitis, intestinal atony of the elderly or debilitated intestinal obstruction, myasthenia gravis, renal function impairment, and ulcerative colitis (severe).

Drug Interactions: MAO inhibitors, Alcohol or CNS depressants, especially anesthetics, barbiturates, and narcotics.

ADVERSE REACTIONS: Prolongs the response to nervous stimulation, potentiates the response to norepinephrine, and inhibits the response to tyramine.

Slight to moderate drowsiness occurs relatively infrequently with Chlorpheniramine Maleate. Other possible side effects common in antihistamines in general include perspiration, chills, dryness of mouth, nose and throat.

Cardiovascular System: Hypotension, headache, palpitations, tachycardia, extrasystoles.

Hematologic System: Hemolytic anemia, thrombocytopenia, agranulocytosis.

Nervous System: Sedation, dizziness, disturbed coordination, fatigue, confusion, restlessness, excitation, nervousness, tremor, irritability, insomnia, euphoria, paresthesias, blurred vision, diplopia, vertigo, tinnitus, acute labyrinthitis histeria, neuritis, convulsions.

Gastrointestinal System: Epigastric distress, anorexia, nausea, vomiting, diarrhea, constipation.

Genitourinary System: Urinary frequency, difficult urination, urinary retention, early menses.

Respiratory System: Thickening of bronchial secretions, tightness of chest and wheezing, nasal stuffiness.

DOSAGE AND ADMINISTRATION: Dosage should be individualized according to the needs and response of the patient. Adults: one capsule every 8 to 12 hours not to exceed 3 capsules daily. Not for use in children under 12 years of age.

OVERDOSAGE: Treatment of the signs and symptoms of overdosage is symptomatic and supportive. In the event of overdosage, emergency treatment should be started immediately.

Treatment: The patient should be induced to vomit, even if emesis has occured spontaneously. Vomiting by the administration of ipecac syrup is a preferred method. However, vomiting should not be induced in patients with impaired consciousness. Stimulants (analeptic agents) should not be used. Vasopressors may be used to treat hypotension. Short-acting barbiturates, diazepam or paraldehyde may be administered to control seizures. Hyperpyrexia, especially in children, may require treatment with tepid water sponge baths or a hypothermic blanket. Apnea is treated with ventilatory support.

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And what is more reassuring to an excessively anxious patient than medication that promptly starts to relieve his discomforting symptoms? Valium® (diazepam/Roche) begins working within 30 to 90 minutes. Patients continue to improve in just a few days, and relief continues throughout the course of treatment.

There are other important benefits with Valium as well—along with its broad clinical range, Valium has an efficacy/safety profile that few, if any, drugs can match. This record has been achieved with extensive clinical experience, undoubtedly including yours. And, as you must have observed, side effects more serious than drowsiness, fatigue or ataxia rarely occur. Nevertheless, as with any CNS-acting agent, patients should be cautioned about driving, operating hazardous machinery or ingesting alcohol or other CNS-depressant drugs while taking Valium.

Yet another benefit Valium affords is flexibility. Available in 2-mg, 5-mg and 10-mg scored tablets, Valium enables you to titrate dosage to individual patient needs. For the geriatric patient, a starting dosage of 2 to 2½ mg once or twice a day is recommended. And, for patients who forget or skip medication, you can prescribe Valrelease™ (diazepam/Roche) 15-mg slow-release capsules, knowing that Valrelease will assure all the benefits of Valium 5 mg *t.i.d.* with the convenience of once-a-day dosage.

Discontinuation of Valium (or Valrelease) is typically as smooth as its start in short-term therapy. However, Valium and Valrelease should be discontinued gradually after more extended treatment. As you diminish dosage, the built-in tapering action of Valium and Valrelease will help avoid rapidly recurring anxiety symptoms and symptoms of withdrawal, and will help ease the patient's transition to independent coping when therapeutic goals have been achieved.

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For a summary of product information, please turn the page.

ROCHE®

Valium® (diazepam/Roche) Ⓝ Tablets
Valrelease™ (diazepam/Roche) Ⓝ slow-release Capsules
Injectable Valium® (diazepam/Roche) Ⓝ

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety. Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic. Symptomatic relief of acute agitation, tremor, impending or acute delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in: relief of skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome. *Oral forms* may be used adjunctively in convulsive disorders, but not as sole therapy. *Injectable form* may also be used adjunctively in: status epilepticus; severe recurrent seizures; tetanus; anxiety, tension or acute stress reactions prior to endoscopic/surgical procedures; cardioversion.

The effectiveness of diazepam in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindications: Tablets or capsules in children under 6 months of age; known hypersensitivity; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence.

> *Usage in Pregnancy:* **Use of minor tranquilizers during first trimester should almost always be avoided because their use is rarely a matter of urgency and because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

ORAL: Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral forms adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures.

INJECTABLE: *To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling and, rarely, vascular impairment when used I.V.: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veins,* i.e., *dorsum of hand or wrist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Injectable Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.*

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea; have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs.

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status. Not recommended for OB use.

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects — particularly with known compounds which may potentiate action of diazepam, *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed and tolerated).

The clearance of diazepam and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

INJECTABLE: Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated.

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug.

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, observed in patients during and after diazepam therapy are of no known significance.

INJECTABLE: Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia.

In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported.

Dosage: Individualize for maximum beneficial effect.

ORAL: *Adults:* Anxiety disorders, relief of symptoms of anxiety—Valium (diazepam/Roche) tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 Valrelease capsules (15 to 30 mg) daily. Acute alcohol withdrawal—tablets, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; or 2 capsules (30 mg) the first 24 hours, then 1 capsule (15 mg) daily as needed. Adjunctively in skeletal muscle spasm—tablets, 2 to 10 mg t.i.d. or q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily. Adjunctively in convulsive disorders—tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily.

Geriatric or debilitated patients: Tablets—2 to 2½ mg 1 or 2 times daily initially, increasing as needed and tolerated (see Precautions). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose.

Children: Tablets—1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use in children under 6 months). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose (not for use in children under 6 months).

INJECTABLE: Usual initial dose in older children and adults is 2 to 20 mg I.M. or I.V., depending on indication and severity. Larger doses may be required in some conditions (tetanus). In acute conditions injection may be repeated within 1 hour, although interval of 3 to 4 hours is usually satisfactory. Lower doses (usually 2 to 5 mg) with slow dosage increase for elderly or debilitated patients and when sedative drugs are added. (See Warnings and Adverse Reactions.)

For dosages in infants and children see below; have resuscitative facilities available.

I.M. use: by deep injection into the muscle.

I.V. use: inject slowly, take at least one minute for each 5 mg (1 ml) given. Do not use small veins, i.e., *dorsum of hand or wrist. Use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.*

Moderate anxiety disorders and symptoms of anxiety, 2 to 5 mg I.M. or I.V., and severe anxiety disorders and symptoms of anxiety, 5 to 10 mg I.M. or I.V., repeat in 3 to 4 hours if necessary; acute alcohol withdrawal, 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary. Muscle spasm, in adults, 5 to 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary (tetanus may require larger doses); in children *administer I.V. slowly*; for tetanus in infants over 30 days of age, 1 to 2 mg I.M. or I.V., repeat every 3 to 4 hours if necessary; in children 5 years or older, 5 to 10 mg repeated every 3 to 4 hours as needed. Respiratory assistance should be available.

Status epilepticus, severe recurrent convulsive seizures (I.V. route preferred), 5 to 10 mg adult dose administered slowly, repeat at 10- to 15-minute intervals up to 30 mg maximum. Repeat in 2 to 4 hours if necessary, keeping in mind possibility of residual active metabolites. Use caution in presence of chronic lung disease or unstable cardiovascular status. Infants (over 30 days) and children (under 5 years), 0.2 to 0.5 mg slowly every 2 to 5 min., up to 5 mg (I.V. preferred). Children 5 years plus, 1 mg every 2 to 5 min., up to 10 mg (slow I.V. preferred); repeat in 2 to 4 hours if needed. EEG monitoring may be helpful.

In endoscopic procedures, titrate I.V. dosage to desired sedative response, generally 10 mg or less but up to 20 mg (if narcotics are omitted) immediately prior to procedure; if I.V. cannot be used, 5 to 10 mg I.M. approximately 30 minutes prior to procedure. As preoperative medication, 10 mg I.M.; in cardioversion, 5 to 15 mg I.V. within 5 to 10 minutes prior to procedure. Once acute symptomatology has been properly controlled with injectable form, patient may be placed on oral form if further treatment is required.

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension. Dialysis is of limited value.

How Supplied:

ORAL: Valium scored tablets — 2 mg, white; 5 mg, yellow; 10 mg, blue — bottles of 100 and 500; Prescription Paks of 50, available in trays of 10; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25 and in boxes containing 10 strips of 10.

Valrelease (diazepam/Roche) slow-release capsules — 15 mg (yellow and blue), bottles of 100; Prescription Paks of 30.

INJECTABLE: Ampuls, 2 ml, boxes of 10; Vials, 10 ml, boxes of 1; Tel-E-Ject® (disposable syringes), 2 ml, boxes of 10. Each ml contains 5 mg diazepam, compounded with 40% propylene glycol, 10% ethyl alcohol, 5% sodium benzoate and benzoic acid as buffers, and 1.5% benzyl alcohol as preservative.

ROCHE®

Dioxin

(Continued from page 529)

decontamination procedure.[13] The half-life of dioxin when incorporated into the soil varies from 200 to 340 days, depending upon soil type and climate. Microbial degradation is probably also responsible for the soil disappearance of TCDD.[11, 13] Uptake of TCDD occurs slowly in plants with no accumulation above external conditions found at maturity.[14]

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Submitted by
Jim Kloeppel, M.S., R.Ph.,
Poison Information Specialist,
Mid America Poison Control Center

References are available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

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References: **1.** Kales A *et al: J Clin Pharmacol 17*:207-213, Apr 1977 and data on file, Hoffmann-La Roche Inc., Nutley, NJ. **2.** Kales A: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **3.** Zimmerman AM: *Curr Ther Res 13*:18-22, Jan 1971. **4.** Kales A *et al: JAMA 241*:1692-1695, Apr 20, 1979. **5.** Kales A, Scharf MB, Kales JD: *Science 201*:1039-1041, Sep 15, 1978. **6.** Kales A *et al: Clin Pharmacol Ther 19*:576-583, May 1976. **7.** Kales A, Kales JD: *Pharmacol Physicians 4*:1-6, Sep 1970. **8.** Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc 27*:541-546, Dec 1979. **9.** Dement WC *et al: Behav Med 5*:25-31, Oct 1978. **10.** Vogel GW: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **11.** Karacan I, Williams RL, Smith JR: The sleep laboratory in the investigation of sleep and sleep disturbances. Scientific exhibit at the 124th annual meeting of the American Psychiatric Association, Washington, DC, May 3-7, 1971. **12.** Pollak CP, McGregor PA, Weitzman ED: The effects of flurazepam on daytime sleep after acute sleep-wake cycle reversal. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, June 30-July 4, 1975. **13.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ.

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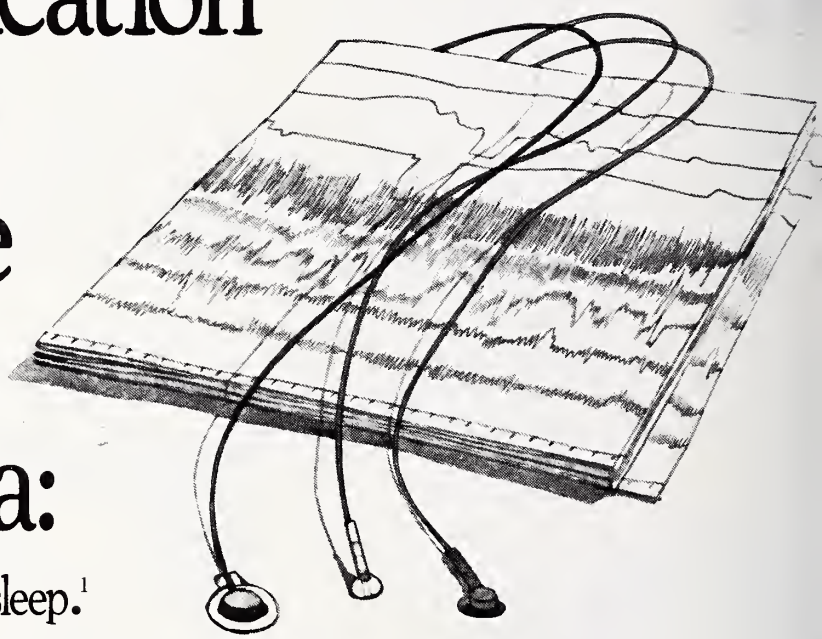
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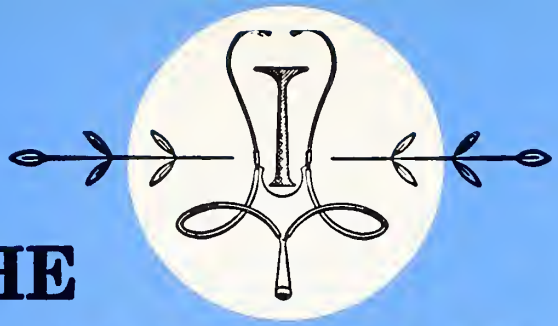
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NO. XI

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Attention of contributors is called to certain policies which the Editorial Board has established in order to meet publication realities. Economic pressures require that, effective immediately, the following policies will be followed in the selection of material for publication:

- Papers will, as always, be reviewed critically for content and professional value and also for economy of space. If reduction of the offering is considered necessary, the author will be notified and given the opportunity to make the necessary alterations as recommended by the Board. Contributors have the option of withdrawing the paper if the Editorial Board's actions are not acceptable.
- The variety of subjects and differing treatments preclude establishing a consistent limitation of length. While economy of space will strongly affect editorial policy, the Board may, at its discretion, accept longer papers. Regardless of article length, contributors have the option of contributing to the cost of printing at the current advertising page rate by mutual agreement with the Board.
- Review articles will not be published. Although some reference to previous material may be necessary, the offering must basically present new material.
- Case reports will be limited to one page or less. Alternatively, brief reports may be submitted as letters to the Editor for consideration for publication in Vox Dox or may, at the discretion of the Editor, be converted to this form.
- Consideration of papers will be limited to those submitted by Kansas contributors or those in which the pertinent material has been developed in Kansas. Exceptions will be at the discretion of the Editorial Board.
- References should accompany the contributions where pertinent in order that the reviewer may have this information. However, they will not be published with the article. Readers will be advised to contact the author(s) for this information.
- Use of graphic material or photographs will be limited to two units per paper. Additional items may be used at the author's expense (actual printing cost).

It is evident that these policies are aimed at the maximum space limitations without reduction in the professional value of the JOURNAL since the ever-increasing publication costs will largely dictate the viability of the JOURNAL.

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The Journal of the Kansas Medical Society

NOTICE

Effective with publication of the December issue of the *Journal*, the following requirements will govern the publication of classified advertising:

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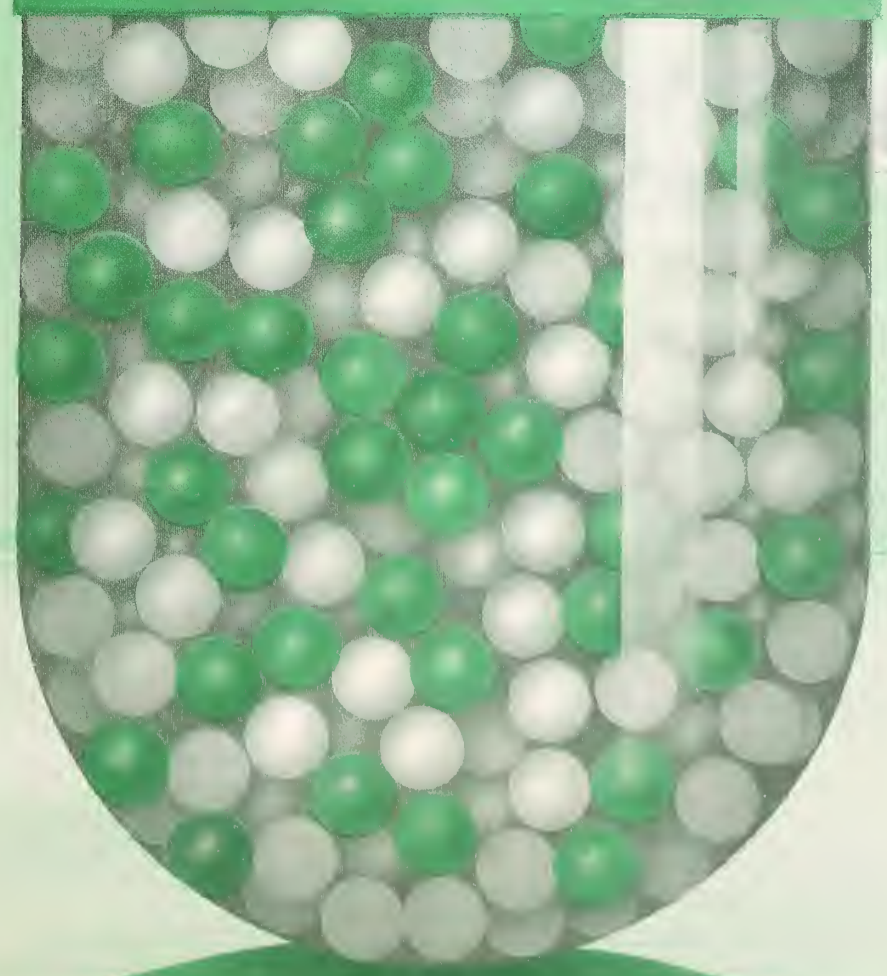
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AUXILIARY NEWS

An Open Letter to Kansas Physicians

Dear Doctor:

Four Kansans joined more than 250 other county presidents elect at the American Medical Association Auxiliary's Leadership Confluence October 9-11 at the Drake Hotel in Chicago. Drunk driving and the prevention of child abuse through effective parenting were among the topics covered in seminars. Other topics included the influence of new technology on children, drug abuse prevention, immunization, and implementing health improvement programs in elementary schools.

Attending from Kansas were: Sherry Hisczynskj (Roman), Shawnee County; Paula Neuer (Fred), Flint Hills; Barbara Ketchum (Lynn), Johnson County; and Terry Yulich (John), Northeast; Diane Sanders (Alan), Lawrence, President; Ann Rempel (John), Wichita, President Elect; Betty Glover (Richard), Newton, Membership Chairman; and Jean Cavanaugh, (Clair), Great Bend, National Director.

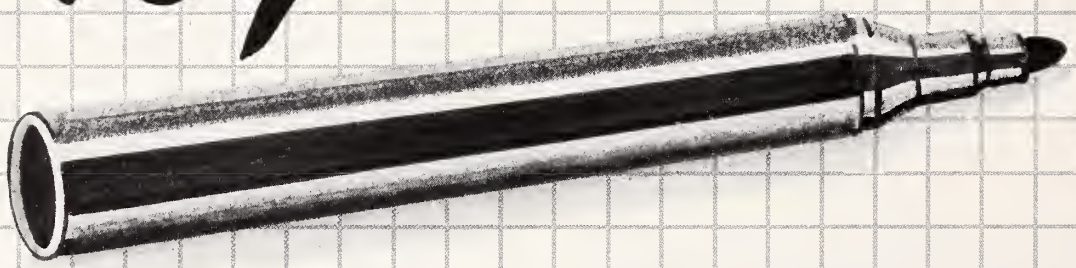
Participants also attended a series of seminars to strengthen leadership abilities in areas such as legislation, parliamentary procedure, organizational financial planning, long- and short-range planning, and communicating orally. Other activities included an early morning walk along the lakefront (President Sanders represented her sleeping delegation), committee meetings and workshops, luncheons, and receptions. Speakers and training films accompanied breakfasts as well as luncheons and dinners — a demonstration of effective time management!

Keynote speaker John J. Coury, M.D., Chairman of the AMA Board of Trustees, discussed DRGs and other elements affecting the current and future practice of medicine. James H. Sammons, M.D., AMA Executive Vice President, brought the confluence to a close at the final luncheon.

The exchange of information on successful community projects and excellent seminars were most enlightening. Our individual reports on the seminars will later appear in the *Kansas Communique*. We sincerely hope that our attendance will enhance the effectiveness of the Kansas Medical Society Auxiliary.

Ann Rempel
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Double Jeopardy

(Continued from page 572)

Answers

1. *False*
2. *True*
3. *False*
4. *False*
5. *False*

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Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety. Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic. Symptomatic relief of acute agitation, tremor, impending or acute delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in: relief of skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome. *Oral forms* may be used adjunctively in convulsive disorders, but not as sole therapy. *Injectable form* may also be used adjunctively in: status epilepticus; severe recurrent seizures; tetanus; anxiety, tension or acute stress reactions prior to endoscopic/surgical procedures; cardioversion.

The effectiveness of diazepam in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindications: Tablets or capsules in children under 6 months of age; known hypersensitivity; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence.

> *Usage in Pregnancy:* **Use of minor tranquilizers during first trimester should almost always be avoided because their use is rarely a matter of urgency and because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

ORAL: Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral forms adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures.

INJECTABLE: *To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling and, rarely, vascular impairment when used I.V.: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veins,* i.e., *dorsum of hand or wrist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Injectable Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.*

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea; have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs.

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status. Not recommended for OB use.

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of diazepam, *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed and tolerated).

The clearance of diazepam and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

INJECTABLE: Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated.

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug.

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, observed in patients during and after diazepam therapy are of no known significance.

INJECTABLE: Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia.

In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported.

Dosage: Individualize for maximum beneficial effect.

ORAL: *Adults:* Anxiety disorders, relief of symptoms of anxiety—Valium (diazepam/Roche) tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 Valrelease capsules (15 to 30 mg) daily. Acute alcohol withdrawal—tablets, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; or 2 capsules (30 mg) the first 24 hours, then 1 capsule (15 mg) daily as needed. Adjunctively in skeletal muscle spasm—tablets, 2 to 10 mg t.i.d. or q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily. Adjunctively in convulsive disorders—tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily.

Geriatric or debilitated patients: Tablets—2 to 2½ mg 1 or 2 times daily initially, increasing as needed and tolerated (see Precautions). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose.

Children: Tablets—1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use in children under 6 months). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose (not for use in children under 6 months).

INJECTABLE: Usual initial dose in older children and adults is 2 to 20 mg I.M. or I.V., depending on indication and severity. Larger doses may be required in some conditions (tetanus). In acute conditions injection may be repeated within 1 hour, although interval of 3 to 4 hours is usually satisfactory. Lower doses (usually 2 to 5 mg) with slow dosage increase for elderly or debilitated patients and when sedative drugs are added. (See Warnings and Adverse Reactions.)

For dosages in infants and children see below; have resuscitative facilities available.

I.M. use: by deep injection into the muscle.

I.V. use: inject slowly, take at least one minute for each 5 mg (1 ml) given. Do not use small veins, i.e., *dorsum of hand or wrist. Use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.*

Moderate anxiety disorders and symptoms of anxiety, 2 to 5 mg I.M. or I.V., and severe anxiety disorders and symptoms of anxiety, 5 to 10 mg I.M. or I.V., repeat in 3 to 4 hours if necessary; acute alcohol withdrawal, 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary. Muscle spasm, in adults, 5 to 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary (tetanus may require larger doses); in children *administer I.V. slowly*; for tetanus in infants over 30 days of age, 1 to 2 mg I.M. or I.V., repeat every 3 to 4 hours if necessary; in children 5 years or older, 5 to 10 mg repeated every 3 to 4 hours as needed. Respiratory assistance should be available.

Status epilepticus, severe recurrent convulsive seizures (I.V. route preferred), 5 to 10 mg adult dose administered slowly, repeat at 10- to 15-minute intervals up to 30 mg maximum. Repeat in 2 to 4 hours if necessary, keeping in mind possibility of residual active metabolites. Use caution in presence of chronic lung disease or unstable cardiovascular status. Infants (over 30 days) and children (under 5 years), 0.2 to 0.5 mg slowly every 2 to 5 min., up to 5 mg (I.V. preferred). Children 5 years plus, 1 mg every 2 to 5 min., up to 10 mg (slow I.V. preferred); repeat in 2 to 4 hours if needed. EEG monitoring may be helpful.

In endoscopic procedures, titrate I.V. dosage to desired sedative response, generally 10 mg or less but up to 20 mg (if narcotics are omitted) immediately prior to procedure; if I.V. cannot be used, 5 to 10 mg I.M. approximately 30 minutes prior to procedure. As preoperative medication, 10 mg I.M.; in cardioversion, 5 to 15 mg I.V. within 5 to 10 minutes prior to procedure. Once acute symptomatology has been properly controlled with injectable form, patient may be placed on oral form if further treatment is required.

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension. Dialysis is of limited value.

How Supplied:

ORAL: Valium scored tablets—2 mg, white; 5 mg, yellow; 10 mg, blue—bottles of 100 and 500; Prescription Paks of 50, available in trays of 10; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25 and in boxes containing 10 strips of 10.

Valrelease (diazepam/Roche) slow-release capsules—15 mg (yellow and blue), bottles of 100; Prescription Paks of 30.

INJECTABLE: Ampuls, 2 ml, boxes of 10; Vials, 10 ml, boxes of 1; Tel-E-Ject® (disposable syringes), 2 ml, boxes of 10. Each ml contains 5 mg diazepam, compounded with 40% propylene glycol, 10% ethyl alcohol, 5% sodium benzoate and benzoic acid as buffers, and 1.5% benzyl alcohol as preservative.

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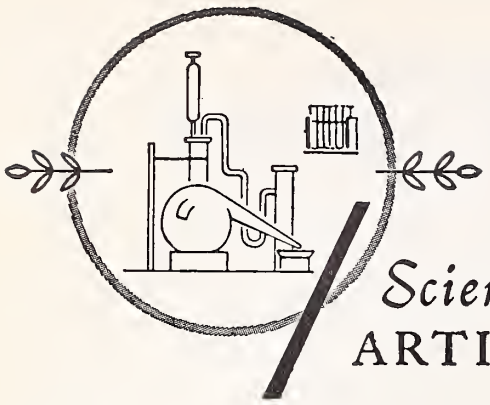
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Endomyocardial Biopsy

Diagnosis of Fabry's Disease in Congestive Cardiomyopathy

PAMELA MABEN, M.D.;* ROGER EVANS, M.D.† *and* JOE LIN, M.D.,‡ *Wichita;*
and* DONNA VAUGHAN, M.D.,§ *Newton

THE CAUSE of congestive cardiomyopathy is unknown in a majority of cases. Conditions identified with congestive cardiomyopathy include: alcohol toxicity, peripartum cardiomyopathy, drug toxicity, infectious and immunologic diseases, and other systemic diseases. Endomyocardial biopsy is usually not helpful in diagnosis and evaluation of congestive cardiomyopathy unless there is a possibility of acute myocarditis, doxorubicin cardiotoxicity, or other systemic disorder.[1]

In the case presented here, acute myocarditis was suspected, but endomyocardial biopsy revealed Fabry's disease.

Case Report

A 43-year-old previously healthy white female presented with a two-week history of progressive dyspnea on exertion, paroxysmal nocturnal dyspnea, and several episodes of short-lived precordial chest pressure not associated with activity. She had had pedal edema for one year, was a smoker, and had been treated for hypertension for five years. The patient's mother had died suddenly in middle-age without previous illness. Her father died of spinal meningitis in his 70s. The patient has four brothers and one sister, all in good health, as well as a son age 12 years and daughter age 16 years, both in good health.

Physical examination showed an obese dyspneic female with blood pressure, 170/110; irregular pulse, 130; and respirations, 28. She had diffuse erythema of the face and arms, three 2-3mm reddish lesions on her back, marked jugular venous distension, rales in the lungs at S_3, and a grade 2/6 systolic murmur at the apex. There was moderate pitting edema of the lower extremities.

Numerous conditions may be identified with congestive cardiomyopathy. In the case presented, endomyocardial biopsy established a diagnosis of Fabry's disease in a patient who presented with a history and findings consistent with an acute myocarditis.

Chest x-ray showed cardiomegaly and increased pulmonary vascularity. Electrocardiogram revealed atrial fibrillation, poor R-wave progression in V_1-V_4, and nonspecific ST-T wave changes. Two-dimensional echocardiogram demonstrated markedly enlarged left atrium and left ventricle. The septum was hypokinetic and was not hypertrophied. The valves appeared normal.

The patient improved somewhat with bedrest, oxygen, diuretics, and digoxin. An endomyocardial biopsy was performed to determine if the patient had an acute myocarditis that might be responsive to steroids and immunosuppressive agents.[1]

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Figure 1. Electron micrograph of heart muscle biopsy showing accumulation of material in the cytoplasm of the cardiac myocytes. This material is a myelin-like substance with concentric laminations and occasional irregular sacs. (X1600)

Endomyocardial biopsy was performed using the Stanford technique from the right internal jugular vein.[1] Light microscopy showed that most of the myocardial cells had fine sarcoplasmic vacuolization and accumulation of foamy material. The material stained pink with methylene blue azure II-basic fuchsin, consistent with glycolipid. Electron microscopy revealed an accumulation of material in the cytoplasm of the cardiac myocytes. This material was a myelin-like substance with concentric lamination and occasional irregular sacs *(Figure 1)*. These findings are diagnostic of Fabry's disease.

The patient continued to improve on only digoxin and diuretics so that one month after biopsy she was in normal sinus rhythm and chest x-ray showed a normal sized heart.

Discussion

Fabry's disease is a rare sex-linked inherited disorder of glycolipid metabolism. There is a deficiency of the lysosomal enzyme alpha-galactosidase A which results in the accumulation of the glycolipid ceramide trihexoside in most tissues of the body but especially skin, kidney, cardiovascular system, liver, spleen, pancreas, and ganglion cells of the peripheral nervous system.[2]

The full expression of Fabry's disease occurs in male hemizygotes. Female heterozygotes usually are asymptomatic or have a limited genetic expression consisting of corneal dystrophy, skin lesions, hypertension, and changes in urine sediment. Rarely, a heterozygote will have a severe expression of the disease with renal or cardiac failure.[3, 4]

The cardiac manifestations of the disease include angina pectoris, myocardial infarction, cardiac hypertrophy and dilatation, and congestive heart failure. All cardiac structures have been found infiltrated with glycolipid including the valves and conduction system.[5, 6]

Treatment for Fabry's disease has been attempted with renal transplantation, enzyme replacement, and plasma exchange with varying success.

Our patient had a severe clinical manifestation of Fabry's disease consisting of congestive heart failure and atrial fibrillation. She had no family history of Fabry's disease. She improved with bedrest, digoxin, and diuretics. Her clinical course was consistent with acute myocarditis, and if she had been empirically started on steroids her improvement and return to normal sinus rhythm would have been attributed to their use. This again emphasizes the usefulness of endomyocardial biopsy when acute myocarditis is suspected and steroid or immunosuppressive therapy is contemplated.[1]

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Acute Extremity Ischemia

Diagnosis and Therapeutic Approach

J. E. BOSILJEVAC, JR., M.D., *Emporia*

THE CURRENT mortality rate for the acutely ischemic extremity remains about 25 per cent.[1, 2] Although generally not appreciated, this high mortality rate is related to the toxins and procoagulants that may be released from the dying tissue, leading to pulmonary and renal problems. Such problems may best be avoided by prompt recognition of the acutely ischemic extremity and immediate management, although an overly aggressive approach in a limb that has suffered from irreversible ischemic damage may also contribute.

Clinical Data

All patients who underwent embolectomy from a prior series[3] with an additional nine patients are listed in *Tables I, II* and *III*. Duration of ischemia is recorded if accurately documented in the chart. These patients were classified according to the degree of ischemia. Degree 1 ischemia refers to patients who had color and temperature demarcation alone. Degree 2 ischemia includes color and temperature demarcation as well as paresthesias or anesthesia but no paralysis. In addition to the above characteristics, patients with Degree 3 ischemia also had paralysis of a major muscle group.

All patients were given a bolus of heparin when initially seen prior to the operative procedure. Embolectomy was performed using a local anesthetic in all cases. Following completion of the embolectomy, an intraoperative arteriogram was done to assess the results. The amputation rate is recorded in *Tables I, II* and *III*, and the mortality rate is summarized in *Table IV*.

Discussion

The high mortality rate for patients undergoing embolectomy is not commonly appreciated. Many of these patients have cardiac and other major system disease, and the high mortality rate may be related to their general medical condition. However, some decrease in the mortality rate may be gained by immediate recognition of the acutely ischemic extremity, and by avoidance of an overly aggressive approach in some patients in whom irreversible damage has occurred.

Presented at the 34th Annual Meeting of the American Heart Association, Kansas Affiliate, July 8, 1983, in Kansas City, Missouri.

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Patients who present with acute extremity ischemia require urgent treatment. Embolectomy is the primary approach; however, considerations for anticoagulation, thrombolytic therapy, or primary amputation are alternatives, depending on clinical status.

When a major arterial obstruction in an extremity occurs, there is an initial protective vasospasm. The vessels distal to the site of obstruction protect themselves from propagation of the clot by this vasospasm. Administration of heparin at this time is protective. As the anoxia in the distal limb persists, the smooth muscle in these vessels relaxes, and the thrombus will propagate into the distal microvasculature. At this point the ischemia is irreversible, and viability cannot be restored by removal of the major obstruction.

The first tissues to be affected by anoxia are the sensory nerves. An initial indication of severe anoxia in an extremity would be the appearance of paresthesias, which then progresses to anesthesia. At this point the situation is still reversible. Next, motor nerves are affected, so paralysis of a major muscle group is recognized. Subsequent to this, patients will begin to suffer irreversible anoxic damage to muscle tissue. The muscles become rigid, edematous, and firm to palpation.

If the obstruction is removed at this point, the anoxic tissue is reperfused and the neurologic symptoms are relieved. If there has been some degree of muscle damage without definite necrosis, there still may be a release of procoagulants and toxins through disrupted cell membranes. These substances may lead to renal failure and pulmonary insufficiency because of their effect on renal tubules and pulmonary surfactant. Cardiac depression may occur, as

TABLE I
PATIENTS WITH DEGREE 1 ISCHEMIA

Age		Level	Degree of Ischemia	Result
78	M	Midcalf	1	Viable
51	F	Midcalf	1	Viable
80	F	Midcalf	1	Viable
69	F	Midcalf	1	Viable
64	M	Midcalf	1	Viable
78	F	Midthigh	1	Viable
57	M	Midthigh	1	Viable
56	F	Midthigh	1	Viable
65	M	Midcalf	1	Viable
55	M	Midcalf	1	Viable
71	M	Midcalf	1	Viable
78	M	Midcalf	1	Viable
67	F	Foot	1	Viable

TABLE III
PATIENTS WITH DEGREE 3 ISCHEMIA

Age		Level	Degree of Ischemia (Duration)	Result
79	F	Midcalf	3 (4 days)	Died
88	F	Midcalf	3 (12 hrs)	Died
56	F	Midcalf	3	Died
68	M	Midthigh	3	Died
87	F	Midcalf	3 (18 hrs)	AK
77	F	Midcalf	3	Died
80	M	Midthigh	3	Died
79	M	Knee	3 (8 hrs)	BK
68	M	Midthigh	3 (4 hrs)	AK
72	M	Midthigh	3 (6 hrs)	AK
80	M	Toes	3 (3 days)	BK
70	M	Midthigh	3 (12 hrs)	AK

well as arrhythmias, not only from these cellular substances, but also from the influx of potassium into the vascular system.

The most important part of the clinical examination is the early identification of an acutely ischemic extremity. Most patients will complain of a rather sudden onset of pain in the extremity. The limb will be cool and pale, or skin mottling may be present. Usually this temperature and color demarcation occurs one joint space below the site of the major obstruction. For example, if the patient was noted to have a color/temperature demarcation at the level of the mid-calf, then one would suspect that the major obstruction was in the superficial femoral artery in the thigh. Pulses will be absent to palpation, and this can be confirmed using a Doppler probe. Next, if paresthesias or anesthesia of the distal extremity is present, this denotes significant anoxia to the sensory nerves. Action of a major muscle group should be examined to see if any paralysis is present, signifying anoxia of the motor neurons. Finally, tenderness and rigidity of the calf musculature indicates severe ischemia with some muscle damage.

At this point, a diagnosis of an acutely ischemic extremity has been made. Most likely this is on the basis of an embolus, particularly if a cardiac source can be identified. Usually these patients will have atrial fibrillation, or will be two or three weeks post myocardial infarction. An embolus may also arise

TABLE II
PATIENTS WITH DEGREE 2 ISCHEMIA

Age		Level	Degree of Ischemia (Duration)	Result
61	M	Midcalf	2	Viable
66	M	Midthigh	2 (4 hrs)	Viable
78	F	Midcalf	2 (5 hrs)	Viable
31	M	Midcalf	2	Viable
69	M	Midcalf	2	Viable
77	F	Midthigh	2 (10 hrs)	Died
53	M	Knee	2 (4 hrs)	BK
66	M	Ankle	2	Viable
58	M	Knee	2 (4 hrs)	Viable
78	F	Midcalf	2 (6 hrs)	Viable
73	F	Midcalf	2 (8 hrs)	Viable
67	M	Foot	2 (10 hrs)	Died
85	M	Midcalf	2 (3 days)	Viable
34	M	Midcalf	2 (4 hrs)	Viable
67	F	Midthigh	2 (4 hrs)	BK
85	F	Foot	2 (3 hrs)	Viable
87	F	Knee	2 (6 hrs)	Died
84	F	Midcalf	2 (2 hrs)	Viable
85	F	Midcalf	2 (2 hrs)	Viable
84	F	Knee	2 (2 hrs)	Viable
70	M	Foot	2 (4 hrs)	Viable

TABLE IV
MORTALITY RATE RELATED TO DEGREE OF ISCHEMIA

	Mortality Rate — Embolectomy	
Degree 1	0/13	(0%)
Degree 2	3/21	(14%)
Degree 3	6/12	(50%)
Overall	9/46	(20%)

from a proximal plaque in the abdominal aorta or a larger proximal vessel in the extremity.

If no cardiac source can be identified, then a thrombus supraimposed on a plaque should be considered. Another clue which indicates that this has occurred would be a history of claudication. Poor pulses in the other extremity would also suggest that significant peripheral vascular disease is already present. Patients who have a thrombus supraimposed upon a plaque in the distal vasculature may tolerate the initial obstruction fairly well if there is a well-developed collateral circulation. Distinguishing between an embolus and a thrombus is important, since a thrombus supraimposed upon an almost obstructing plaque in the distal vasculature will not be well treated by embolectomy. A major revascularization or bypass procedure may be necessary in this situation.

Another consideration in the acutely ischemic extremity would be atherosclerotic emboli (blue toe syndrome). This may occur as a macroembolus, which is similar to a major obstruction as previously described. Microembolization from an atherosclerotic plaque may also occur *(Figure 1)*. This may consist of platelet aggregates or clots which form on the ulcerated surface of a plaque and embolize distally. Atherosclerotic material or pieces of the plaque itself may also embolize. These patients generally will present with ischemia or gangrene of a digit, such as a toe, or with arterial ulcerations on the distal extremity. They may also have some mottling or discoloration of the skin (livedo reticularis). However, on examination they will be noted to have fairly good peripheral pulses. Evidence of acute ischemia in the presence of palpable pedal pulses suggests microembolization of atherosclerotic material (blue toe syndrome). Despite the fact that the patients have palpable pedal pulses, they are still at risk for limb loss if treatment is not undertaken. Approach to the blue toe syndrome has been discussed in a prior paper.[1]

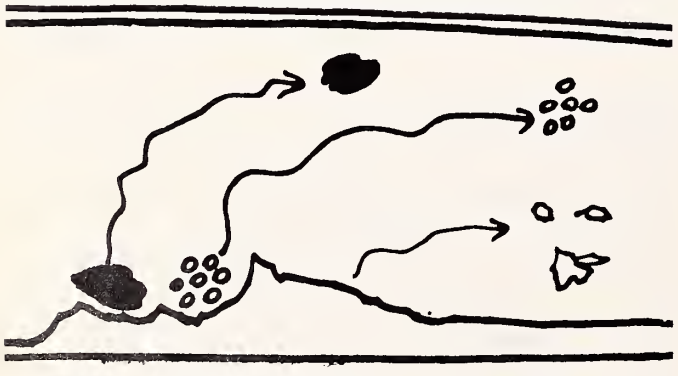


Figure 1. Embolization from a proximal plaque may consist of thrombus, platelet aggregates, or atherosclerotic material.

Diagnosis of an embolus *vs* thrombus *vs* an atherosclerotic embolus should be accurate in a range of about 90 per cent from history and physical examination. The major consideration at this point would be: Is limb-threatening ischemia present? If the patient has paresthesias or anesthesia or paralysis of a major muscle group, then these patients are in the category of Degree 2 or 3 ischemia. As can be noted from *Tables II* and *III*, patients with this degree of ischemia are at high risk for limb loss. The mortality rate is higher in these groups also.

With regard to current therapeutic approach, embolectomy is still the primary treatment.[4-6] Prior to embolectomy, the patient should be given a large bolus of heparin to prevent propagation of thrombus in the distal microvasculature. The embolectomy is usually performed using a local anesthetic, which makes the procedure safer for these elderly patients who may have other concurrent major system disease. Intraoperative arteriograms should be performed to evaluate the vascular tree following embolectomy.

In patients who show evidence of muscle damage (Degree 3 ischemia) and who would not survive pulmonary or renal complications, primary amputation may be the treatment of choice.[2] Also, if the patient's cardiac status is poor and s/he could not tolerate congestive heart failure or further insult following the release of procoagulants or toxins from ischemic tissue, an amputation may be considered. In the presence of anesthesia, paralysis of a major muscle group and any evidence of calf tenderness or rigidity indicating major muscle damage, caution should be used before embarking on an aggressive approach.

High dose heparin[2, 7, 8] as well as streptokinase (thrombolytic) therapy[9, 10] has been recommended by some as initial treatment for the acutely ischemic extremity. These may be an alternative if the patient is at high risk for surgical therapy, although embolectomy performed under a local anesthetic is usually well tolerated in elderly patients. Medical therapy should be considered only in patients who have a viable limb on initial examination. Also, patients who have peripheral vascular disease that is beyond arterial reconstruction may be given a course of thrombolytic treatment prior to amputation, if no definite gangrene is present. However, even small doses of thrombolytic agents injected intra-arterially

(Continued on page 575)

Harrington Rod

Instrumentation and Fusion of Unstable Spinal Fractures

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SURGICAL STABILIZATION of unstable fractures of the thoracic and lumbar spine has received considerable attention during the past two decades. Although advocates of treatment by recumbency report satisfactory results,[1] this method is expensive, time-consuming, and requires specialized nursing care. In 1958, Harrington first utilized his instrumentation system (developed for scoliosis) in the management of an unstable spinal fracture.[2, 3] This method of treatment has subsequently gained increasing popularity.[4-6] The application of Harrington distraction rods helps achieve the immediate goals of stabilization of the fracture, decompression of the neural elements, and restoration of spinal alignment. With these goals achieved, early institution of a rehabilitation program is made possible.

Unstable fractures of the thoracic and lumbar spine can be successfully treated by recumbency. However, use of the Harrington instrumentation system can achieve satisfactory results at lower cost and afford early initiation of a rehabilitation program.

Materials

Between November 1976 and December 1982, a total of 78 patients with unstable spinal fractures underwent instrumentation and fusion. The patient population consisted of 59 males and 19 females with an average age of 29.5 years (range: 14 to 62 years). Distribution of the level of fractures is shown in *Figure 1*. The mechanisms of injury are shown in *Table I*. There was complete paraplegia in 26 (30%) of the patients *(Table II)*. Associated injuries are listed in *Table III*. Pre-operative evaluation included anteroposterior and lateral roentgenograms of the spine. During the early part of this series, tomograms were frequently used to evaluate the fracture. Considerably better visualization of the injured area became available with the institution of computerized axial tomography (CAT) studies.[7] Myelography was not routinely performed.

Methods

All patients underwent stabilization with two Harrington distraction rods. The upper hooks were implanted two intact lamina above the fracture site and the lower hooks were implanted one or two intact lamina below the fracture site. Autogenous iliac crest bone was employed in fusing the spine from the upper to the lower hook levels. Decompressive laminectomy was performed only in incompletely paralyzed patients whose CAT studies revealed significant bony encroachment into the spinal canal which would not be relieved by restoration of spinal alignment alone. Fourteen patients met this criteria and underwent laminectomy.

Postoperatively, the patient was placed on a standard hospital bed. Log-rolling was performed every two hours. Between the third and fifth postoperative day, a removable Plastazote body jacket was molded for the patient. Mobilization was then begun on a tilt-table with progression to a wheelchair or to ambulation. The use of the body jacket was continued for approximately six months, until roentgenograms revealed a solid fusion.

Results

There was one death in the series which occurred in a patient with severe head trauma. No patient with complete paraplegia showed any degree of neurologic recovery. No patient with incomplete paraplegia became worse, and most showed neurologic improvement. One patient experienced hook displacement with loss of fixation which required reoperation. One patient with burns developed a wound dehiscence which required secondary closure. Three patients had documented pulmonary emboli. There were no deep wound infections. A total of four patients subsequently underwent rod removal (one year or more postoperatively) for painful bursa over the upper tip of the rods.

Summary

The Harrington instrumentation system provides an effective method of stabilization of thoracic and

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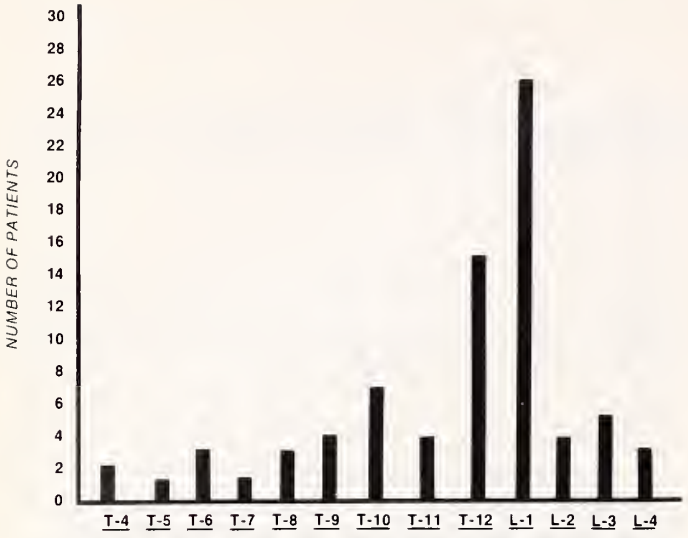


Figure 1. Distribution of level of fracture among patients in this series.

lumbar spine fractures. Restoration of spinal alignment — with laminectomy in selected cases — allows decompression of the neural elements. Early institution of a rehabilitation program reduces post-op complications, decreases hospital stay, and promotes the earlier achievement of the patient's full rehabilitation potential.

Acknowledgments

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TABLE I
CAUSE OF ACCIDENTS

Automobile	41
Motorcycle	7
Fall	15
Struck by object	12
Aircraft	3

TABLE II
NEUROLOGIC STATUS

Complete paraplegia	26
Incomplete paraplegia	37
Neurologically intact	15

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TABLE III
ADDITIONAL INJURIES

None	62
Head injury	1
Long bone fractures	11
Chest injury	3
Abdominal/pelvic	1
Burns	3
Amputation (traumatic)	1
Lacerations requiring surgery	2
Knee ligament damage	1

Biostator Glucose Controller

Its Use During Surgical Removal of an Insulinoma

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SINCE WILDER's classic descriptive report of a functioning islet cell carcinoma,[1] hyperinsulinism has become a suspected diagnosis in patients exhibiting a fasting blood glucose of 50 mg/dl or less. Whipple set forth "Whipple's Triad"[2] consisting of hypoglycemic symptoms in the presence of hypoglycemia (a blood glucose level less than 50 mg/dl), which are relieved by the ingestion of glucose. The diagnosis of insulinoma (hypoglycemic syndrome) can be further clarified today by obtaining immunoreactive insulin (IRI) levels in conjunction with blood glucose levels. A high ratio of plasma insulin to glucose (greater than 0.3) is considered a reliable diagnostic feature.[3]

The most common of the entopically functioning islet cell tumors responsible for hypoglycemic syndrome is benign insulinoma. It constitutes approximately 70-80 per cent of insulin secreting tumors as a singly occurring adenoma. Multiple or malignant tumors and hyperplasia constitute the remaining percentage. This large percentage of benignity found in entopic lesions is in marked contrast to those found ectopically.[3]

Several provocative tests are available to assist in making the diagnosis of insulinoma.[3-6] After diagnosis, the tumor must be delineated. This has been reliably accomplished in approximately 90 per cent of cases by angiography.[7] Computerized tomography of the pancreas has also been reported as a helpful localization procedure.[4]

The greatest difficulty of the surgical procedure lies in decision making regarding pancreatic resection. If a single tumor is located and excised, is it the only tumor or are there multiple lesions? Or, is the hyperinsulinemia due to islet cell hyperplasia instead of tumors? If so, how much of the pancreas should be excised for optimal functioning? Often blood glucose monitoring is used as a surgical adjunct to detect whether or not all tumors have been removed, or that adequate pancreatic resection has been achieved. Literature reports that blood glucose may drop drastically upon palpation of the tumor. A sharp hyperglycemic rebound has been noted within 30 minutes after removal of hyperfunctioning islet cell tissue. This rebound is usually inhibited or completely negated if more than one lesion is present or if excess hyperplastic islet cell tissue remains *in situ*.[8] It is in the area of blood glucose monitoring and control that the Biostator glucose controller has proven to be highly beneficial both diagnostically and for prevention of hypoglycemia during surgical operations.

Unrecognized hypoglycemia in an anesthetized patient can complicate pancreatic surgery. The case presented here demonstrates the effectiveness of the Biostator glucose controller for monitoring such patients and controlling glucose levels during surgery.

The Biostator

The Biostator glucose controller is a machine used to monitor and control blood glucose. The system involves passing a constant flow (approximately 2 cc/hr) of the patient's blood across a membrane-bound enzymatic glucose sensor. A special double lumen catheter dilutes and anticoagulates the blood as it leaves the patient. The blood glucose level is displayed continuously while six values per minute are averaged, with the average printed on a permanent record. A preprogrammed computer calculates a dose of glucose or insulin according to the blood glucose level. This dosage is then administered intravenously to the patient via a peristaltic pump system. Use of the system has been reported in further detail in current literature.[9-11]

Case Report

A 19-year-old woman had been under treatment for a "seizure disorder" since six years of age. These "seizures" were characterized by "fading spells" and periods of unconsciousness. Just prior to

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these spells the patient reported feeling lightheaded, numb, and hungry, with chills and sweating. These "seizures" were relatively well-controlled by anticonvulsant medications and a six-meal plan until approximately 14 months prior to admission. At that time the patient began to experience frequent episodes of blacking out, accompanied by tonic/clonic seizure activity with complete amnesia. Her medication was evaluated and changed, and she was referred to a psychiatrist. Shortly after this the patient's mother found her unconscious and unarousable in bed. She contacted the patient's psychiatrist who arranged for hospital admission. Upon admission the patient had a blood glucose level of 18 mg/dl.

After admission, a glucose tolerance test was performed and the patient was found to be hypoglycemic with a preliminary fasting blood glucose of 22 mg/dl in the presence of an IRI of 50 μU/ml. The entire six-hour glucose tolerance test showed an abnormally high insulin level per glucose level and a hypoglycemic nadir at six hours. Computerized tomography and sonography of the abdomen showed no definitive lesion; however, arteriography showed a hypervascular mass in the distal body of the pancreas. The patient was prepared for surgery using the Biostator for blood glucose monitoring and control.

On the day of the operation, the patient was attached to the Biostator glucose controller. Her blood glucose level was set in the range of 40-60 mg/dl. The surgical procedure began with the patient's blood glucose controlled at 48 mg/dl. At 10:15 A.M., a tumor was removed that, upon pathologic examination, proved to be a benign islet cell adenoma. Within 15 minutes the blood glucose level began to rise in the absence of Biostator glucose administration, at which time insulin administration per Biostator increased *(Figure 1)*. Biostator control was discontinued at 11:10, with continuation of blood glucose monitoring for continued assurance that a second tumor had not been overlooked; at this point the operation was concluded. At 1:21 P.M., the patient's blood glucose reached 278 mg/dl and Biostator control was resumed; however, the glucose sensing membrane required replacement at 2:00, causing a brief interruption of glucose and insulin infusion. Biostator monitoring and control was discontinued at 5:30 after blood glucose stabilization in the range of 100-120 mg/dl was achieved during a one-hour period. Fasting blood glucose levels were drawn each postoperative morning with normalization occurring on the fifth postoperative day *(Figure 2)*.

Discussion

Observation of the endogenous insulin and glucose levels, and insulin given by the Biostator as shown in *Figure 1*, reveals several interesting occurrences. The endogenous glucose level was initially quite low as anticipated due to overnight patient fasting; the endogenous insulin level was not exaggerated but still inappropriately high for the level of glucose. As preparation for the operation and induction of anesthesia began, blood glucose

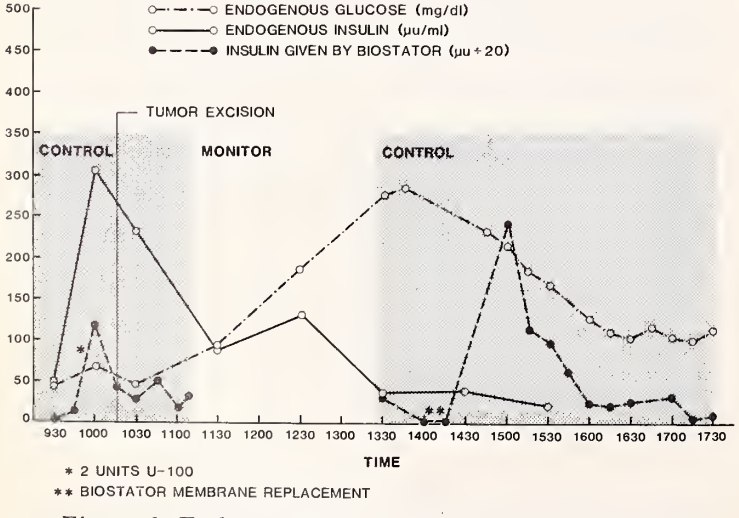


Figure 1. Endogenous insulin and glucose levels, and insulin dosage given by the Biostator on the day of the operation.

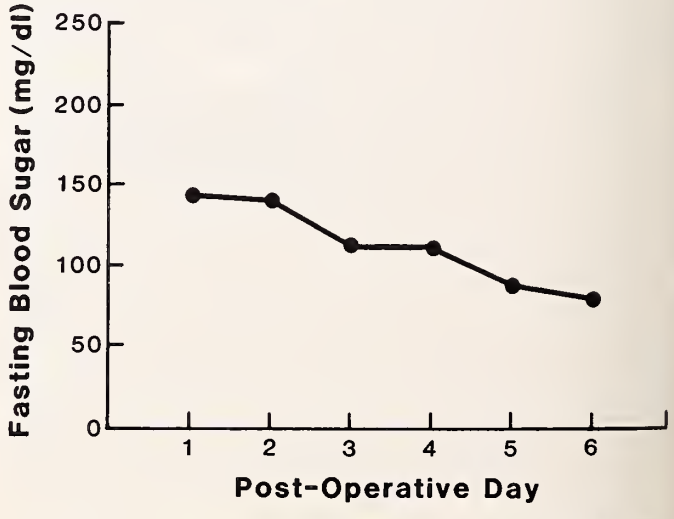


Figure 2. Daily postoperative fasting blood glucose levels.

rose slightly causing an abrupt increase in endogenous insulin secretion. The Biostator also reacted to the rising blood glucose by increasing insulin administration, although not as extreme as endogenous levels. During a 15-minute interval (9:45-10:00) the machine administered approximately two units of U-100 insulin (2,293 μU). As the blood glucose leveled and dropped, the Biostator responded immediately by decreasing insulin administration.

During the period of Biostator monitoring without control following tumor excision (11:10-1:21), blood glucose increased appropriately as expected (hyperglycemic rebound after tumor excision).[8] Of interest is that endogenous insulin levels continued to fall despite rising blood glucose. This can be interpreted as a combination of two occurrences. It may reflect the fact that the only insulinoma had been completely excised (none retained in the patient). It may also be that the non-responsiveness of remaining beta cells to rising glucose levels is due to their prior suppression from the hypersecreting insulinoma. It has been reported by Liljenquist[12] that exogenous insulin administration may suppress the normal pancreatic insulin release. This would appear to hold true for hypersecretion of endogenous insulin. The latter phenomenon is similarly and commonly observed after successful excision of a single parathyroid adenoma in sporadic hyperparathyroidism. In the case of hyperparathyroidism, remaining parathyroid glands are, and have been hypofunctioning and thus do not respond immediately to the falling serum calcium levels.

The large peak of insulin seen occurring from 4:15 to 5:00 following resumption of Biostator control, is an appropriate response of five units to a blood glucose above the normal level. As the patient's blood glucose normalized into a less jagged graphic representation, so did the Biostator insulin administration. By 5:30, the need for insulin was minimal when blood glucose normalized at homeostatic levels.

Summary

Reports and discussion of the hypoglycemic syndrome due to hyperinsulinemia are commonly found in surgery and endocrinology literature. The surgical exploration and excision of insulin-secreting tumors or hyperactive islet cell tissue may be complicated by the possibility of unrecognized hypoglycemia in an anesthetized patient. These potential problems can be effectively monitored and controlled by use of the Biostator glucose controller. The Biostator can be used in several ways for this type of surgery, one of which was described in this case report. In this case, the patient's blood glucose was controlled at a slightly hypoglycemic state until excision of the tumor. The blood glucose was then monitored until it attained a significant hyperglycemic rebound, at which time control was resumed until blood glucose stabilization was achieved. By use of the Biostator, the surgeon had continuous knowledge of the blood glucose level as an adjunct to decision making regarding tissue excision. The possibility of extreme hypoglycemia during the surgical operation was negated by Biostator blood glucose control. The Biostator has been shown to be a useful and efficient adjunct to surgical correction of the hypoglycemic syndrome.

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Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Double-Jeopardy — Cancer in Old Age

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CANCER is very much a disease of the elderly; cancer incidence increases almost logarithmically after age 40 years, with one-half of all cancers diagnosed after age 65 years, although those over 65 comprise only 12 percent of the population. Two reasons are prolonged exposure to cancer-inducing agents, and waning power of immune defenses against cancer.

Two possible objectives in treatment of cancer are cure or control. For both, the best measure of success is prolongation of life. In order to establish that a method of cancer treatment is better than no treatment at all, or that one method is better than another, one must consider the survival experience of scores or hundreds of patients to achieve significance by statistical analysis. Survival analysis for cancer patients can be done by a number of different methods, most of which seem to have been derived from the modification of actuarial analysis described by Berkson and Gage.[1] Comparisons can easily be made to survival curves for the population at large for various ages, and the excess mortality from the cancer is apparent.

For purposes of treatment planning and for prognosis, the stage or extent of spread of a cancer at the time of diagnosis is a very important determination. There are many schemes for staging cancer, some quite complex. However, the common denominator is a four-level system described in *Table I*. After site of origin, stage at diagnosis is the most important determinant of survival. There is no consistent relationship between stage of cancer at diagnosis and age at diagnosis, although older people are more likely to have advanced cancers at diagnosis than are younger people.[2] In that expected survival duration decreases as one considers ever older segments of the general population, it becomes apparent that age is a doubly-critical factor in cancer. Increasing age is associated with ever-increasing cancer incidence but also with ever-decreasing life expectancy.

Both cancer and its active treatment cause pain and discomfort in addition to raising the specter of death. A child with cancer is usually in otherwise good health and, if cured, presumably has a life expectancy of many decades. In the older patient, who may be frail and suffering from several chronic diseases, and whose life expectancy is a relatively few years, there is much less justification for aggressive treatment that will cause discomfort and that may even shorten a life with but a few years to run at best. In the elderly with cancer, we must be certain that we do not make the treatment worse than the disease. We must have a clear idea of risks and benefits of various treatments and be prepared to accept limited goals. We must often "temper the wind to the shorn lamb."

Methods

Incidence and survival data are presented for two cancers common in the elderly — prostate in men and breast in women. *Table II* shows incidence data for these two cancers. *Table I* shows the definition of stages used for the survival information plotted in *Figures 1* to *6* for Kansas patients diagnosed between 1950 and 1979. The six figures show actuarial survival for a stage (local, regional, or distant) of a site (prostate or breast) for three age groups (65-74, 75-84, and 85-94 yrs). Expected survival for that age group in Kansas from 1970 census data is plotted in the stippled area, females at the top border and males at the bottom border. The solid line denotes survival

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TABLE I
STAGE OF CANCER AT DIAGNOSIS

Stage	*Extent of Disease*
In Situ (0)	Confined to surface of site of origin
Local (I)	Confined to organ or tissue of origin
Regional (II)	Spread to area around organ of origin or to regional lymph nodes
Distant (III)	Spread to place(s) distant from site of origin

of those patients diagnosed between 1970 and 1979, and the broken line for those diagnosed between 1950 and 1969. The actuarial method of survival calculation is that of Berkson and Gage, and takes into account observed mortality, not relative mortality.[1]

Prostate

The most prevalent cancer affecting elderly men in the United States originates in the prostate gland. With average age at diagnosis slightly exceeding 72 years, it is the quintessential cancer of old age. As shown in *Table II,* incidence increases inexorably with age, reaching 1,061/100,000 men age 85 years or older.

There are two landmarks in the history of treatment of prostate cancer. In 1966 Charles Huggins was awarded the Nobel Prize in Medicine for his 1941 description of the effects of castration and estrogens on prostate cancer and the implied benefits to patients.[3] Ironically, in 1967 Arduino *et al.*[4] presented data from the first controlled trial of treatments for prostate cancer. Arduino and associates established conclusively that there was no difference in survival duration in respect to treatment. They proscribed castration for reasons other than symptom relief in advanced disease and noted, "what estrogen treatment wins from the cancer, it more than loses to other causes of death."

Chemotherapy in prostate cancer has demonstrated no clear survival advantage to date according to Torti and Carter.[5] Radiation — particularly interstitial radiation — has been the most useful form of treatment for prolonging survival, a fact long known but insufficiently appreciated.

From 1950 to 1979 there was no significant improvement in the survival rate in prostate cancer staged as local at diagnosis *(Figure 1).* Its contribution to mortality for the three age groups lessens until it is inconsequential in the group 85 to 94 years old at time of diagnosis. The majority of these patients are treated surgically, with radiation of various types being used more frequently in recent years. The survival curves in *Figure 1* clearly show that prostate cancer of this stage is usually an indolent disease. The concept of a five-year cure is meaningless; in fact, many believe that this disease is rarely if ever cured at any stage. In the age groups presented here

TABLE II
AGE-SPECIFIC INCIDENCE OF BREAST CANCER IN FEMALES AND PROSTATE CANCER IN MALES EXPRESSED AS CASES PER 100,000 POPULATION OF THAT SEX BY FIVE-YEAR AGE GROUPS

Age	*Breast*	*Prostate*
35-39	57	—
40-44	106	1
45-49	174	5
50-54	196	21
55-59	229	68
60-64	251	166
65-69	283	319
70-74	302	514
75-79	338	735
80-84	350	961
85+	376	1,061

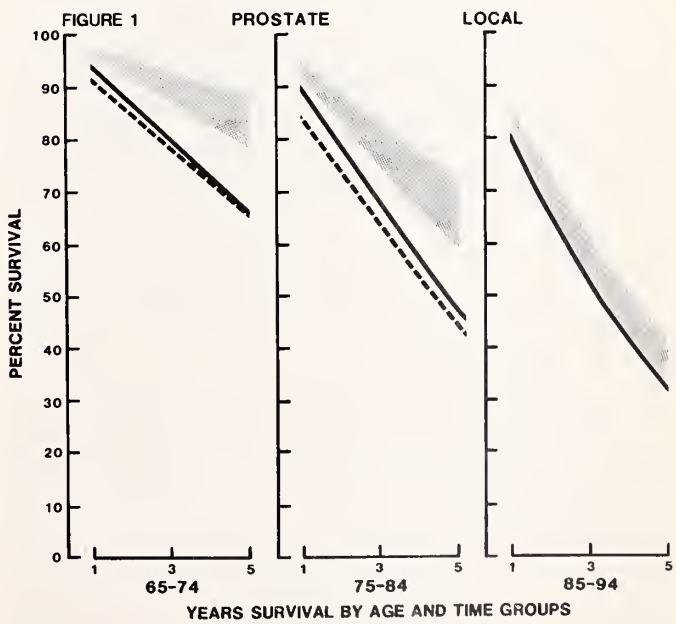


Figure 1. Surivival rates for patients with prostate cancer staged as local at diagnosis. Solid line, 1970-1979; broken line, 1950-1969; stippled area, expected survival in Kansas from 1970 census data.

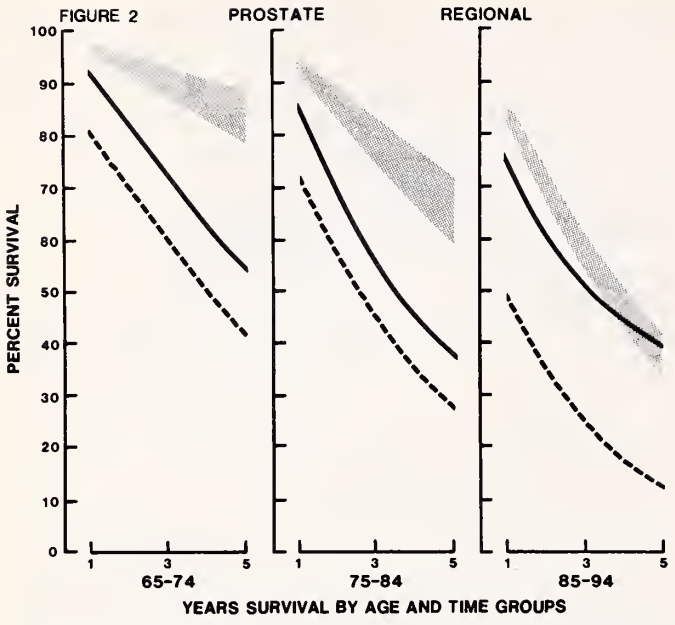


Figure 2. Survival rates for patients with regionally spread prostate cancer at diagnosis. Solid line, 1970-1979; broken line, 1950-1969; stippled area, expected survival in Kansas from 1970 census data.

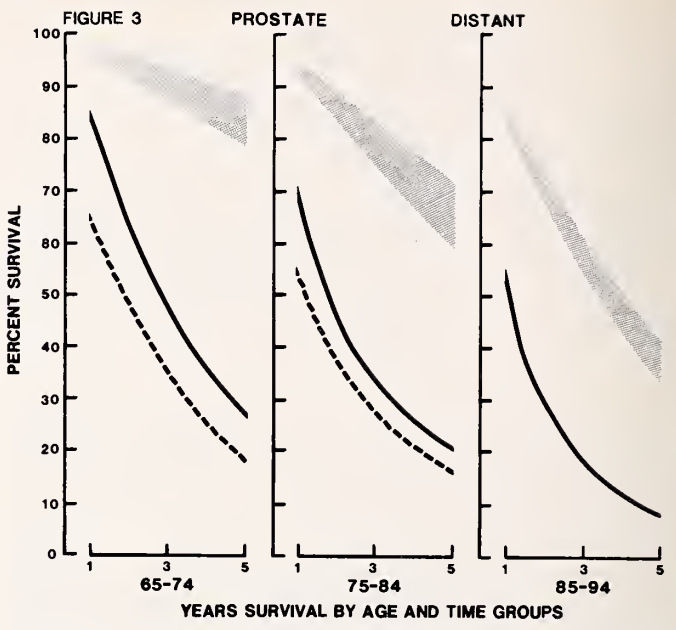


Figure 3. Survival rates for patients with widespread prostate cancer at diagnosis. Solid line, 1970-1979; broken line, 1950-1969; stippled area, expected survival in Kansas from 1970 census data.

there is no justification for mutilation or radical treatment as an attempt to cure a few patients. About one-half of these patients die with evidence of persistence of prostate cancer, and one-half have no obvious evidence of it at death. The most sensible management strategy would seem to be control of disease manifestations, including early identification of disease recurrence or progression.

Good news can be derived from 30-year data for regionally spread prostate cancer *(Figure 2)*. There is statistically significant increase in the survival rate at one, two, and four years for the 65-74 age group; one and two years for the 75-84 age group; and at five years for the 85-94 age group. There are at least two reasons for this. In the 1970s, estrogen was used less frequently and, when used, in much smaller amounts than in the 1950s and 1960s. External beam and interstitial radiation have been more extensively used in recent years.

The great irony of prostate cancer treatment is clearly shown in the 85-94 age group. The 1970s survival nearly equals that of the general age-matched male population, a vast improvement since the 1950-1969 period. In fact, there is little ground left to gain at this age. Regionally spread prostate cancer can be said to have minimal influence on mortality in this group of the old now that estrogen treatment has been appropriately reduced.

Curves from the two younger age groups show that there is still excess mortality that might be reduced, but not much. The best treatment strategy would seem to be an aggressive effort to control the cancer in the pelvis at the time of diagnosis, with early identification of disease progression or recurrence, and appropriate control measures then applied. Given the limits of surgery in this regard and the futility of chemotherapy as presently practiced in this disease, a major role for radiotherapy is suggested.

Prostate cancer widespread at diagnosis is not easily treated; it is a systemic disease and taxes the skill of any physician. Because extensive bony metastases are the rule at this stage, the patient is often disabled by bone pain. The curves in *Figure 3* show an improved survival rate during the 30 years of the study, but it is significant only at one year in the 65-74 age group. Likely, modification of estrogen doses is as much responsible for this modest improvement as any other factor. Of course, virtually all patients with this stage of disease die *with* the disease but not always *of* it.

Surgery and radiation play limited roles at this stage of the disease. This group of patients needs effective systemic therapy — probably chemotherapy or something closely akin to it. Unfortunately, nothing appropriate and effective is presently available. However, with control of symptoms — particularly bone pain — it is remarkable how many men with this stage of disease may have years of comfortable and useful life.

Breast

Although breast cancer is the most common cause of death for middle-aged women in America, it is not often appreciated that the incidence rate climbs steadily into old age as shown in *Table II*. In 1894 Halstead described radical mastectomy in 50 patients, and this treatment for breast cancer remained essentially unquestioned until challenged by Crile in 1974.[6,7] The best operation for breast cancer is still uncertain. Beyond this there is considerable feeling that radiation of early breast cancer may be just as effective as surgery.[8] Finally, to complete the confusion, the utility of chemotherapy as adjuvant treatment following surgery to prevent recurrence is now fairly well established.[9]

Breast cancer is a capricious disease, chronic for some women and acute for others. Even cursory inspection of survival curves shows that five-year disease-free survival does not mean cure as it does in many kinds of cancer. In fact the record for initial treatment for cure to recurrence is 50 years with intervals of 20 years or so sufficiently common that they are not reportable.[10]

These factors have special relevance for older women with breast cancer. Minimal rather than heroic and mutilating surgery seems quite appropriate for the elderly. Long-term complications of radiation and chemotherapy have little relevance in older patients. Thus, if radiation of limited breast cancer is as good as surgery, it is an excellent choice for the elderly woman. When life expectancy is measured in years rather than in decades, early recurrence of breast cancer becomes much more important than late recurrence. Adjuvant chemotherapy seems to be particularly efficacious for elderly women if its benefits are clearly established and if accompanying discomfort and inconvenience are less than the disability of recurrence.[11]

Survival in breast cancer confined to the breast at diagnosis is surprisingly good. In viewing *Figure 4*, one must remember to compare the two survival curves to the top margin of the gray area. Most women with this stage of disease are cured by initial treatment — usually surgery. Unfortunately, these data cannot be used to answer questions of how much surgery is enough or whether radiation is as good as surgery in treating this stage of disease.

Figure 4 shows no significant or even apparent increase in survival in the 30 years of this study. Excess mortality, in the youngest age group particularly, is from early progression of distantly spread disease not detected at the time of diagnosis and from late recurrence. Interestingly, breast cancer *per se* exerts little influence on mortality in the two older

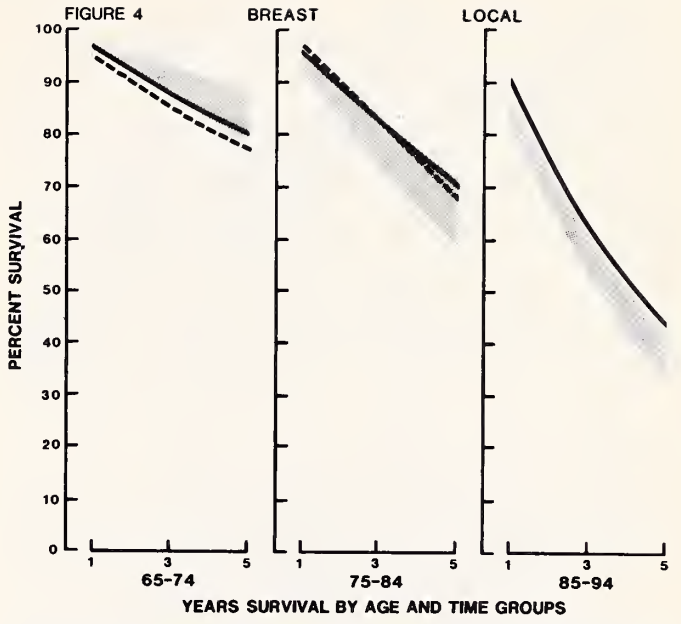


Figure 4. Survival rates for patients with breast cancer staged as local at diagnosis. Solid line, 1970-1979; broken line, 1950-1969; stippled area, expected survival in Kansas from 1970 census data.

age groups, most deaths being from other causes, although early progression is a factor of importance and worth recognition.

The challenge in this stage of disease is to prevent, or at least delay, early progression of distantly spread undetected disease. Chemotherapy adjuvant to potentially curative therapy has been proposed as the appropriate strategy. Unfortunately, one has to accept the fact that most women so treated are already cured and are, thus, needlessly exposed to the dangers and discomforts of chemotherapy. In the 1950-1969 period, there were 212 women with locally confined breast cancer at diagnosis who were between 65 and 74 years of age. Of these, 65 (30.6%) died with breast cancer — 15 (7.1%) ten or more years after diagnosis. A woman in the 65-74 age group has, on the average, a life expectancy of 15 years. She has much to gain if adjuvant chemotherapy works. At least in regionally spread disease, it seems to delay early disease progression and perhaps increases survival duration. Long-term complications of adjuvant chemotherapy have yet to be defined, but may well include increased risk of second cancers, particularly acute leukemia. It will take some years to learn whether or not adjuvant chemotherapy will alter patterns of later recurrence of breast cancer. This is an important factor for women in the 65-74 age group although of relatively less importance for those in the two older age groups. Analysis of this stage of disease indicates

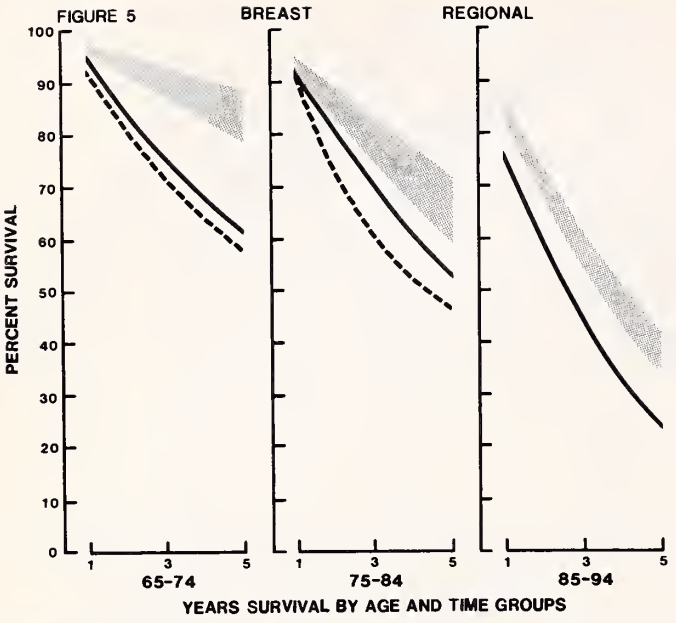


Figure 5. Survival rates for patients with regionally spread breast cancer at diagnosis. Solid line, 1970-1979; broken line, 1950-1969; stippled area, expected survival in Kansas from 1970 census data.

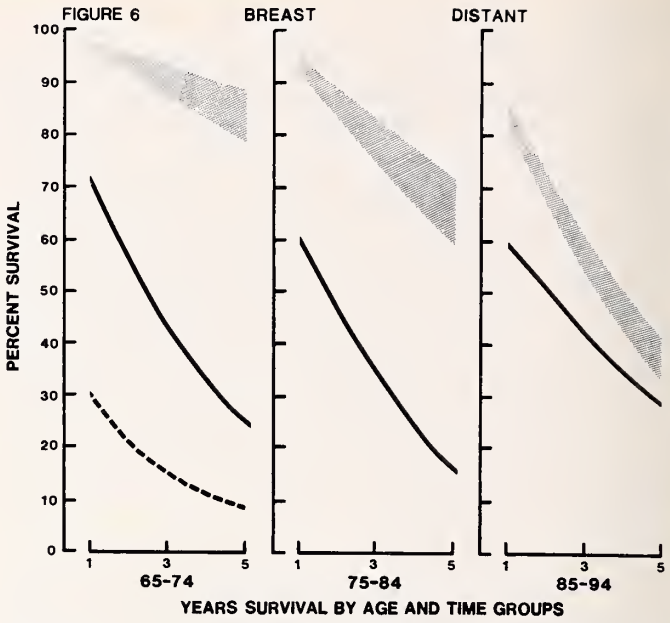


Figure 6. Survival rates for patients with widespread breast cancer at diagnosis. Solid line, 1970-1979; broken line, 1950-1969; stippled area, expected survival in Kansas from 1970 census data.

that locally confined breast cancer is a disease usually and easily cured for most women but likely to become a chronic disease for those not cured.

Regional spread of this disease means spread of the tumor to lymphatic tissue, into the chest wall, or into the skin overlying the breast. *Figure 5* shows modest apparent improvement in survival during the 30 years of this study, but this is not statistically significant. Excess mortality from breast cancer is apparent in all three age groups. More than one-half of women with breast cancer in this stage at diagnosis eventually die with or of their disease, as data from the 1950-1969 period prove (115 of 216 dead with disease, 32 dead more than five years after diagnosis). Thus, a sizable minority of women with breast cancer at this stage are probably cured, but for most it is a chronic disease.

For all age groups in this study, prevention or delay of early progression of disease is the most important goal. Late recurrence is still of some importance, particularly for the 65-74 age group, but its prevention or further delay is much less important than earlier progression. Thus chemotherapy, adjuvant to initial treatment, assumes a high order of importance.

All women with breast cancer at this stage at time of diagnosis die of or with their disease. *Figure 6* shows the profound mortality rate effect in all three age groups. At least in the 65-74 age group, survival is significantly increased at one, two and three years. The only plausible explanation for this is the efficacy of multi-agent chemotherapy protocols used extensively during the 1970s. It may also be true for the two older groups, but numbers of cases in the 1950-1969 time period were too small for meaningful analysis in this study.

Again it is surprising that for some women this is a chronic disease even when diagnosed at this advanced stage. The actuarial uncorrected five-year survival rate is 23.3 per cent in the 65-74 year-old age group in the 1970s. Occasionally chemotherapy or hormonal therapy produces fairly long periods of apparent disease-free remission even at this stage. The challenge for management of this stage of breast cancer is control of disease principally by systemic therapy. Past successes would seem to indicate that more effective multi-agent protocols can be devised.

Other Cancers

Although lung cancer incidence rates decline after about age 75 years, rates at younger ages in both sexes continue to climb. Lung cancer is curable at any age but only in a small percentage of those diagnosed, and this percentage has not changed in 30 years. Perhaps better control should be our immediate goal in dealing with lung cancer. The significant survival improvement at one and two years in regionally spread disease and lengthened survival in

small cell histology may be the heralds of this. Although normal lung tissue tolerates radiation poorly, it seems likely that improved radiotherapy techniques will facilitate disease control and short-term survival still further. The big question is whether or not effective chemotherapy can be devised to deal with non-small cell histologies. This would greatly facilitate control of incurable disease, and might improve survival rates after potentially curative surgery as it has in breast cancer.

Considered collectively, gastrointestinal cancers are far and away the major force in cancer morbidity and mortality in the aged in America. It is ironic indeed that as incidence of stomach cancer declines, pancreas cancer inexorably increases, and colorectal cancer is stable although there is more right-side colon cancer and less rectal cancer. We have little understanding of etiologic factors in these disease entities. The irony is compounded by the fact that the efficiency of our treatment efforts remains essentially unchanged, either cure or failure, with control barely a consideration. The exception is a significantly improved one-year survival rate in regionally spread stomach cancer in age group 75-84. In this regard it can be stated that, at any age, stomach cancer is cured rarely, pancreas cancer virtually never, and colorectal cancer in about one-half of cases. Surgery is the only effective therapy; chemotherapy and radiotherapy have been of little use.

Gynecologic cancer would extract little mortality if every woman, regardless of age, had an annual pelvic examination and pap smear. The fact that virtually all women now regard post-menopausal vaginal bleeding as abnormal and quickly seek medical advice when it occurs is probably the reason that endometrial cancer is usually diagnosed at an early stage and usually cured. Unfortunately, cervix and ovarian cancers make their presence known later in the course of the disease, and cure rates are correspondingly lower.

Widespread endometrial and cervical cancers are essentially untreatable diseases by current methods of therapy, with chemotherapy of little use. When these two diseases are regionally spread at diagnosis, radiotherapy gives relatively good cure rates and excellent control of disease not cured. Although ovarian cancer is not diagnosed at an early stage as often as it should be, surgical cure rates are excellent. Both radiotherapy and chemotherapy make significant contributions to control of regional and widespread disease, and there is every reason to believe that modifications of these modalities will improve disease control in the future.

In contrast to prostate cancer, both kidney and bladder cancer are usually not indolent diseases but are eminently curable when diagnosed at an early stage. Long survival — say greater than four years — usually means cure. Kidney cancer cure rates are excellent for locally confined disease and fair for regionally spread disease, while bladder cancer cure rates are even better. Early diagnosis is, thus, the key to effective treatment. Radiotherapy is supplemental to surgery in bladder cancer and useful for palliation in kidney cancer. Chemotherapy is very disappointing in both.

Collectively the hematologic malignancies are a major cancer morbidity and mortality force in old age. They range from the most acute to the most indolent of cancers. Some older patients with Hodgkin's disease and histiocytic lymphoma may be curable, but as a general rule it can be stated that hematologic cancers in the elderly are incurable. During the last ten years, there has been remarkable improvement in survival in aged patients diagnosed with plasma cell myeloma and the lymphomas. This improvement is doubtless due to the development of effective chemotherapy well beyond the improvement in survival one might expect from better supportive care. In stark contrast are the leukemias where no significant survival improvement has been noted in spite of intensive efforts to perfect multi-agent chemotherapy and in spite of greatly improved supportive care. The contrast within the leukemias is great also. The acute leukemias extract maximum mortality, and chronic lymphocytic leukemia extracts little mortality — treatment offering little or nothing in either instance. For all of the hematologic malignancies, future improvement in survival will devolve on better chemotherapy or discovery of entirely new treatment methods. For the present, appropriate treatment of the elderly patient with a hematologic malignancy demands a thoughtful and careful physician.

In conclusion, one might observe that the secrets of both aging and cancer and then of increased cancer with increased age are probably locked deep in the nuclei of cells in DNA itself. Ultimately we may be able to devise more efficient methods of cancer treatment, and these may well be made possible by advances in molecular biology. Until these improvements in understanding and therapy, we are best advised to keep in focus the finitude of human life and the natural histories of various kinds of cancers. Then balancing benefit and risk to each patient, particularly the elderly, we will be better physicians.

Self-Assessment Questions

1. Even regionally spread prostate cancer can be surgically cured in elderly men. (T or F)
2. Multi-agent chemotherapy lengthens survival in elderly women with breast cancer widespread at time of diagnosis. (T or F)
3. Survival time for elderly patients with acute leukemia has substantially increased in the past 30 years. (T or F)
4. Alkylating agents significantly increase survival in chronic lymphocytic leukemia in the elderly. (T or F)
5. Lung cancer is more common in 80-year-olds than in 70-year-olds. (T or F)

(Answers on page 551)

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The President's Message

Economic issues continue to be a source of frustration and concern. Implementation of many new phases in economic policies will begin shortly.

Each of you has made a decision regarding the Blue Cross/Blue Shield contracts. The decision to sign or not to sign was a painful experience. I fear we will have many other difficult decisions to make in the future. We must make our decisions on the basis of continued excellent quality of service, our patients' needs, and our professional intent.

Professional liability problems continue to increase. The recent Kansas State Supreme Court ruling about open records related to hospital settings is very disturbing. Physicians must be able to continue their peer review, utilization review, and quality assurance programs. The professional liability committees of the KMS and the Kansas Hospital Association will work very hard to seek legislative changes in these laws.

The cost of professional liability protection continues to increase. The KMS is working diligently with the Insurance Commissioner's office to formulate revisions in the law so that costs may be analyzed more appropriately for all of us.



All of us must be continuously involved in these decisions. The KMS needs your input as we continue to strive to be the aggressive organization we need and must be in these times.

Jeannee A. Reasoner M.D.

President



No single thing abides, but all things flow.
Fragment to fragment clings — the things thus grow
Until we know and name them. By degrees
They melt, and are no more the things we know.
—Lucretius

Some future Garrison, seeking to update that classical medical history, may well conclude that a major shift in medical function was born in the last quarter of the 20th century. If he (or she) avoids the urge to relate it to the amazing expansion of medical technology or the upheaval in ethical concepts which accompanied it (and did indeed contribute greatly to that shift), the resulting record should read that it was the force of economic, not academic, pressures that altered the medical character. But it must be remembered that this historian will be looking back after the fact and, being acclimated to a different medical world, may not recognize the contemporary impact of such force on the medical profession and its individual components.

"Profession." The word itself holds a key. Its offspring, "professional," holds particular interest not because it serves as both noun and adjective — many words do that — but because its usage has come to emphasize divergent meanings. Traditionally, it has defined that class of individuals set apart by advanced learning or intellect — applied in general to many disciplines but to none more aptly or characteristically than medicine. But at some point, the word acquired a secondary meaning, that payment for the services of such persons was a qualifying feature. Not surprisingly, in a materialistic world, this identification with making money has become dominant in the public mind and, by extension, is considered mainly to differentiate it from amateur. Thus, common usage has a way of debasing lofty enterprises and, in this case, leads to neglect of the important characteristics of professionalism.

First, there is the dedication of the participants to individual standards of ability and integrity, imparting to the group its collective distinction. Second, however, it is explicit in this distinction that it should survive by self-regulation based on the acknowledgement that those within the profession have the capacity, perspective, and dedication to judge their own professional propriety and moral soundness in assuring effective application and control.

Such is the picture of ideal professional function in an ideal society. Since both entities fall somewhat short of the ideal, society has increasingly declared its dominance by imposing its own regulations — sometimes in support, sometimes in conflict with those the profession finds essential. The result has been that, in the name of public necessity, society's regulations have increasingly superceded professional scruples. This encroachment of public regulation is, furthermore, self-begetting. Each new regulation not only nullifies some of the autonomy of the profession but, in the view of zealous public servants, demonstrates the need for controls in other directions.

To put this consideration of medical professionalism on hold for the moment and look at the medical scene from a different angle, 1983 can be recorded as the time when the complex factors relating to medical costs came to a sort of fruition in a bevy of plans to "control" those costs. Although numerous organizations (including some sponsored by physicians) offered their particular versions of the True Way, the basic feature is the same: financial remuneration for services would be predetermined and fixed. Since payment for the largest portion of medical care has moved progressively into the hands of various agencies — social, commercial, governmental — their decisions have the effect of economic (and sometimes public) law. The stated intent of these plans is to create a climate of competition among the providers of medical care, thus achieving economic stringency by controlling and regulating the remuneration. Initially, there has been little frank statement that physicians' fees will be deliberately set and controlled (more than they have been by previous fiat), but the element of competitive bidding has been officially established and physicians will have the choice of conforming to competition or suffering exclusion from the favored circle. At the same time, there has been pressure to bring physicians into service categories where physician remuneration (not to mention service methods and conditions) will be established by the remunerative administrations.

It is apparent that these actions will bring a spate of regulations — they live by them — and while such regulations are by no means new, they will now be all-enveloping. Although the stated intent is fiscal and general, the effect will be professional and personal. The various agencies will, of course, justify such intrusions as good spirited and intended only for the benefit of all. The word of the day is that it has to be — and there doesn't seem to be much of the camel left outside. The glimpse we get of medical professionalism of the future is not encouraging. Physicians will, as always, be able to effect a degree of professionalism in their direct contacts with individual patients but increasingly they will have to do it against regulatory strictures and in a climate that will tend to stifle those intrinsic qualities of professionalism to which physicians have always subscribed.

How much professionalism will "suffer" depends upon one's viewpoint. The idea implies that the concept as we have known it will be forced into a new form. Rather, we think, it will be a different professionalism, not without some of the qualities of its predecessors but retaining less of the traditional and conforming more to a "new wave" (read commercialized) professionalism. Ours is an interesting age and, at risk of inciting outcries from knowledgeable historians, we believe we are seeing for the first time a major medical movement effected by social forces. This is not to overlook the changes in both the profession and society brought about by the medical "miracles" of this century, although society's adjustment (or lack of it) has contributed significantly to the current conditions. Moreover, we admit that we are imparting to the matter a form and effect that owes a certain amount to editorial rhetoric. Nevertheless, the traditional structures — hospitals, practice methods, academic systems, physician relationships with each other and with their support personnel — all are being subjected to fiscal pressures that can only lead to increasing controls which cannot help but produce a new professionalism.

As our historian should know if he (or, again, she) is serious about the craft, the past is replete with social changes wrought by medical events — the caravans that took occidental diseases to the Orient and brought oriental diseases back, the travelers who traded in gold and silk as well as worms and germs. There is no better example than the Black Plague of the 15th century which, in the process of destroying some 20 per cent of the population, brought social impact which we are still feeling 500 years later. But she (or is it he?) may, with the proper perspective, see that in this event, the tables are turned: medical professionalism is being determined by social dicta.

The process will not be immediate and therefore, at any given moment, there may be morose assessments such as this, but there will also be professional acceptance and adaptation — ranging from reluctant to avid. As the current crop of elderly curmudgeons moves out of the way, the system will be assimilated by those coming on — even perhaps, being defended as a better and more enlightened (therefore more professional) way. But if these comments come to light in some archeological dig of the future, let our historian (him or her — we really don't care) know that the age produced a certain amount of dismay among some of the professional ancestors. — *D.E.G.*

Acute Ischemia Extremity

(Continued from page 560)

can markedly deplete fibrinogen and cause bleeding problems should surgery become necessary later.

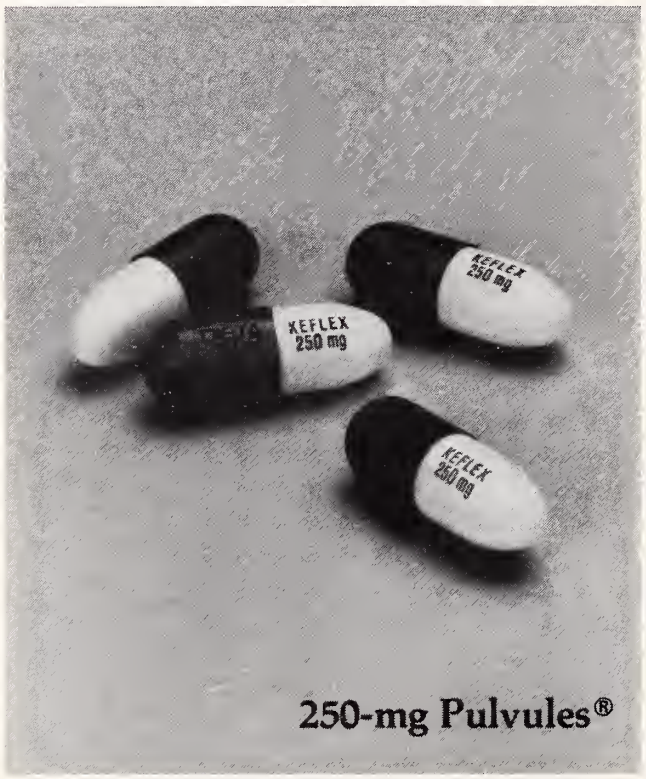
Summary

The mortality rate for the acutely ischemic extremity remains in the range of 25 per cent despite modern advance in medical and surgical therapy. Early diagnosis is extremely important in limb salvage, and differentiating an embolus from a thrombus or atherosclerotic embolus may alter the approach. Primary amputation in patients who have evidence of irreversible ischemia may trade limb loss for life loss in some cases.

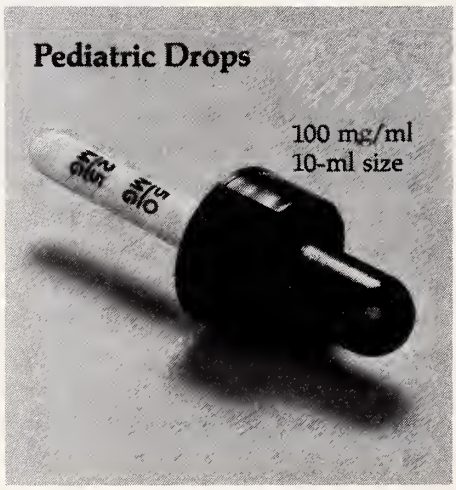
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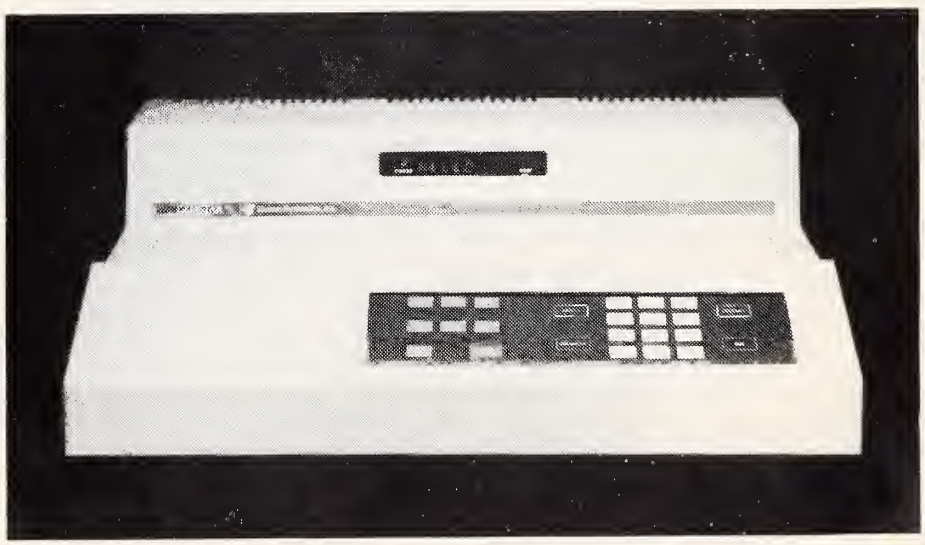
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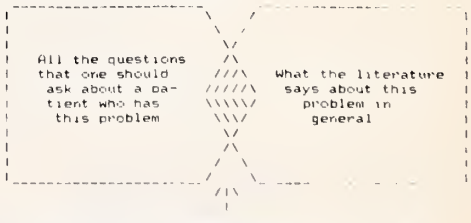
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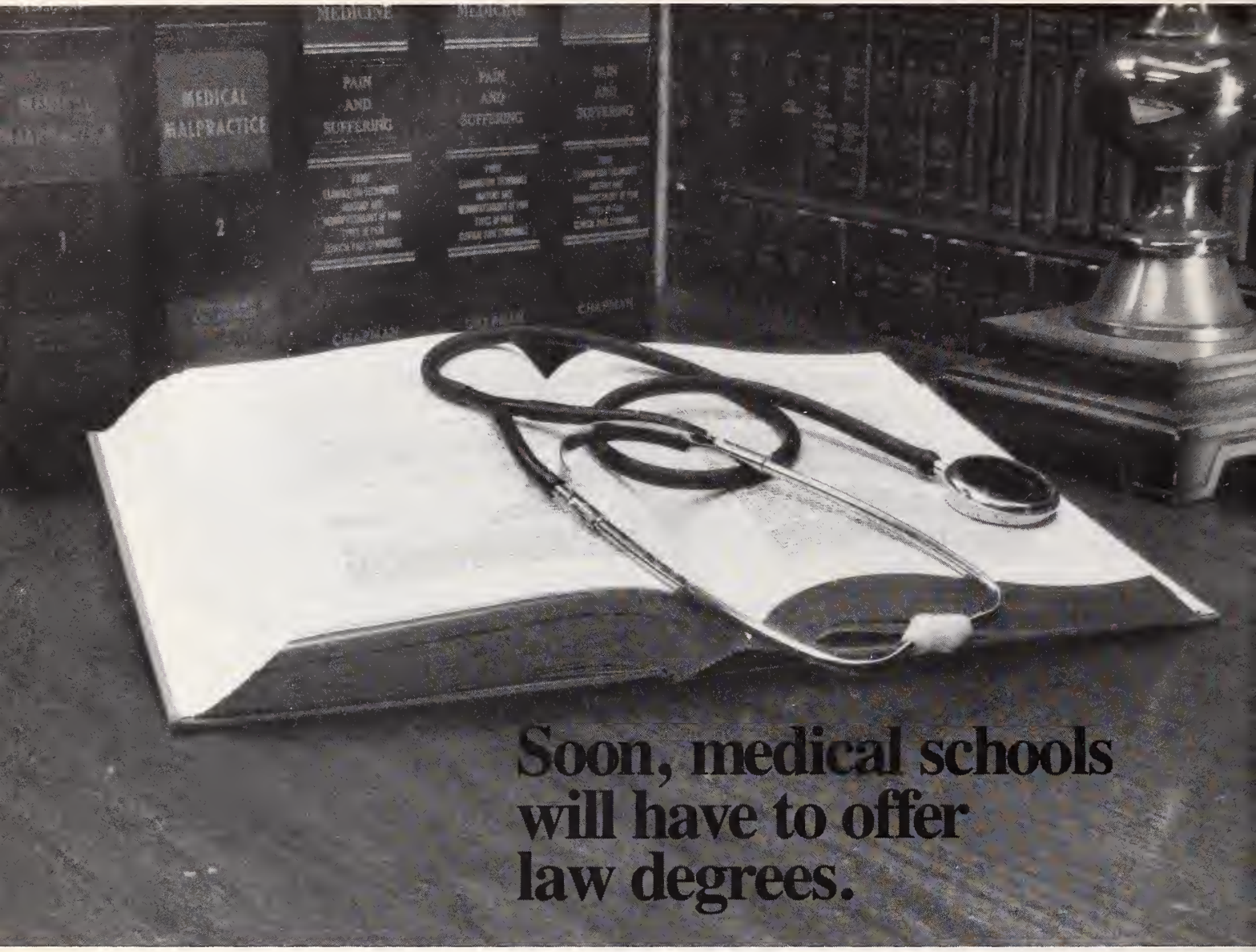
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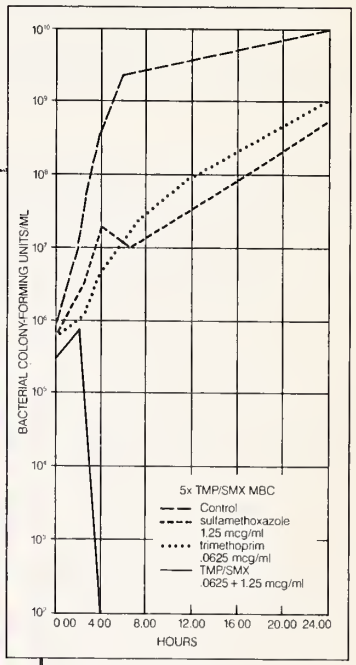
Bactericidal activity with minimal resistance

Percent of isolates of common uropathogens sensitive to BACTRIM and to other antimicrobials

	Escherichia coli	*Klebsiella pneumoniae*	*Proteus mirabilis*	*Proteus vulgaris*	*Proteus sp.*	*Enterobacter sp.*	*Enterobacter aerogenes*	*Enterobacter cloacae*
BACTRIM	**96%** 328,598	**89%** 63,551	**93%** 62,691	**84%** 4174	**91%** 4381	**88%** 1736	**96%** 7326	**92%** 11,856
ampicillin	72% 345,209	5% 65,466	85% 67,338	16% 4381	70% 4402	10% 1775	9% 7759	11% 12,091
cephalexin†	81% 343,339	85% 67,031	92% 67,027	16% 4398	80% 4365	22% 1834	13% 7571	12% 11,846
nitrofur-antoin	97% 307,785	65% 61,162	7% 59,775	13% 3848	14% 3059	67% 1707	60% 7109	67% 10,830

†Analogous to cephalothin, the primary antibiotic disc used in testing.
Source: The Bacteriologic Report, BAC-DATA Medical Information Systems, Inc., Winter Series, 1981-82.
Numbers under percentages refer to the projected number of isolates tested.

RAPID *IN VITRO* DESTRUCTION OF *E. COLI* *



Kill curve kinetics of Bactrim and its individual components against *E. coli in vitro.*[1]

The bactericidal action of Bactrim has been demonstrated *in vitro* on laboratory strains of *E. coli*[1,2] and on clinical isolates of *E. coli, Klebsiella-Enterobacter, Proteus mirabilis* and *Morganella morganii*[3]—the most common causative organisms of urinary tract infections.[4] More than 100 published studies attest to the efficacy of Bactrim in recurrent urinary tract infections due to these organisms.[5] In comparative studies with other antimicrobials, Bactrim has consistently demonstrated unsurpassed efficacy during therapy.[6-11]

Resistance to Bactrim develops more slowly than to either of its components alone *in vitro.** Among urinary tract isolates, resistance has rarely emerged in susceptible strains.[5,12] Bactrim is contraindicated in pregnancy at term, during lactation, in infants less than two months old and in documented megaloblastic anemia due to folate deficiency.

Initial episodes of uncomplicated urinary infections should be treated with a single-agent antimicrobial.

Bactrim™ DS

(trimethoprim and sulfamethoxazole/Roche)

b.i.d. *for recurrent urinary tract infections*

**In vitro* data do not necessarily predict clinical results.

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See next page for references and a summary of product information.

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Bactrim™ DS

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii*. It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, hepatocellular necrosis, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients. *Pregnancy:* Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, hepatocellular necrosis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: *Not recommended for infants less than two months of age.*

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:

Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS:

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets*, each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 20. *Tablets*, each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. *Pediatric Suspension*, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz (1 pint). *Suspension*, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per tea spoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).

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Nutley, New Jersey 07110

References:
1. Stone PH, Turi ZG, Muller JE: Efficacy of nifedipine therapy for refractory angina pectoris. *Am Heart J* 104:672-681, September 1982
2. Antman E, Muller J, Goldberg S, et al: Nifedipine therapy for coronary-artery spasm: Experience in 127 patients. *N Engl J Med* 302:1269-1273, June 5, 1980.

BRIEF SUMMARY

PROCARDIA® (nifedipine) **CAPSULES** For Oral Use

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents: (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients; transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

More detailed professional information available on request

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"I can do things that I couldn't do for 3 yrs. including joining the human race again."



Quotes from an unsolicited letter received by Pfizer from an angina patient. While this patient's experience is representative of many unsolicited comments received, not all patients will respond to Procardia nor will they all respond to the same degree.

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"My daily routine consisted of sitting in my chair trying to stay alive."

"My doctor switched me to PROCARDIA[*] *as soon as it became available. The change in my condition is remarkable."*

"I shop, cook and can plant flowers again."

"I have been able to do volunteer work...and feel needed and useful once again."

PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks,[1] taking fewer nitroglycerin tablets,[2] doing more, and being more productive once again.

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%).



for the varied faces of angina

PROCARDIA®
(NIFEDIPINE) *Capsules 10 mg*

*Procardia is indicated for the management of:
1) Confirmed vasospastic angina.
2) Angina where the clinical presentation suggests a possible vasospastic component.
3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

Please see PROCARDIA *brief summary on adjoining page*

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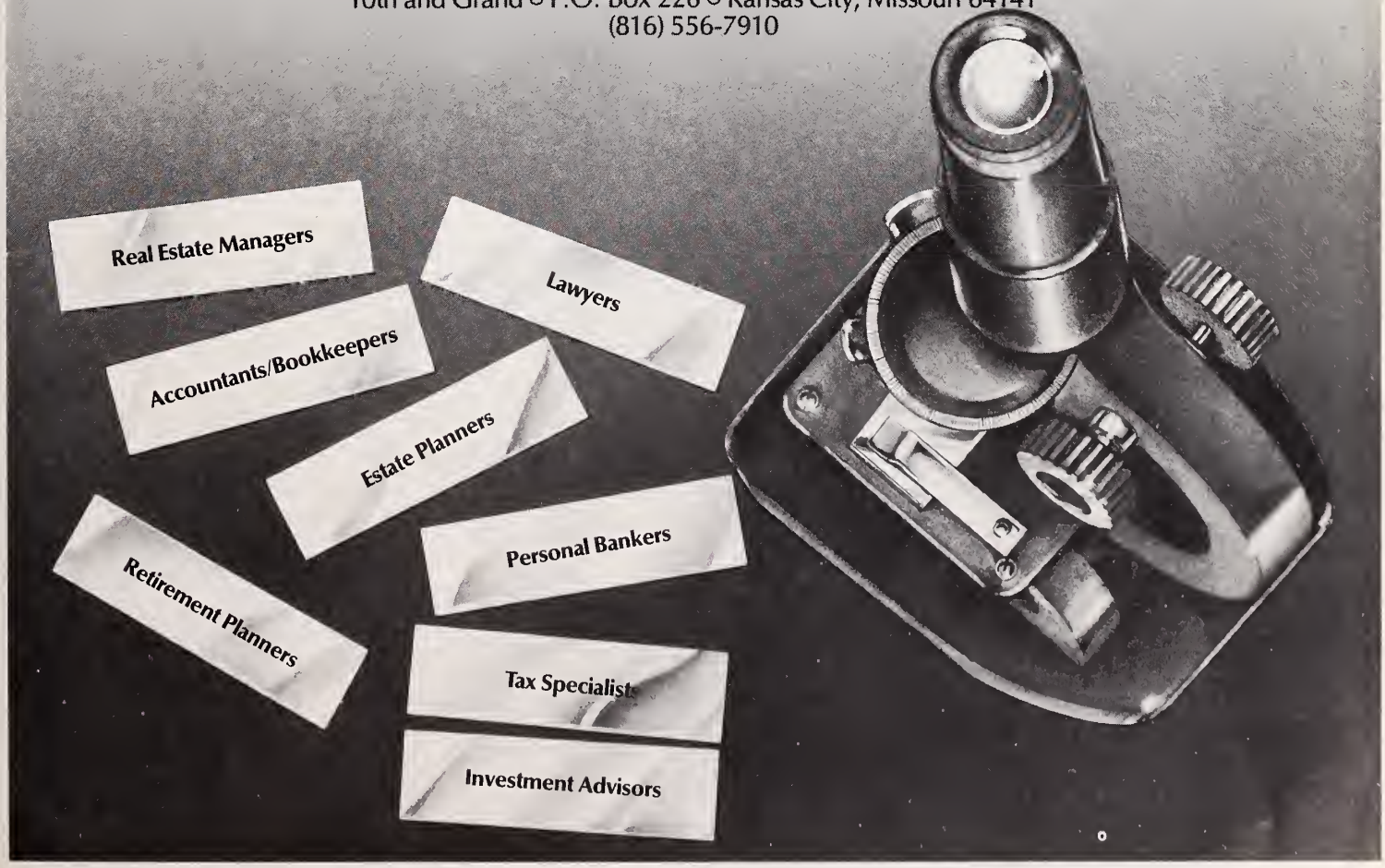
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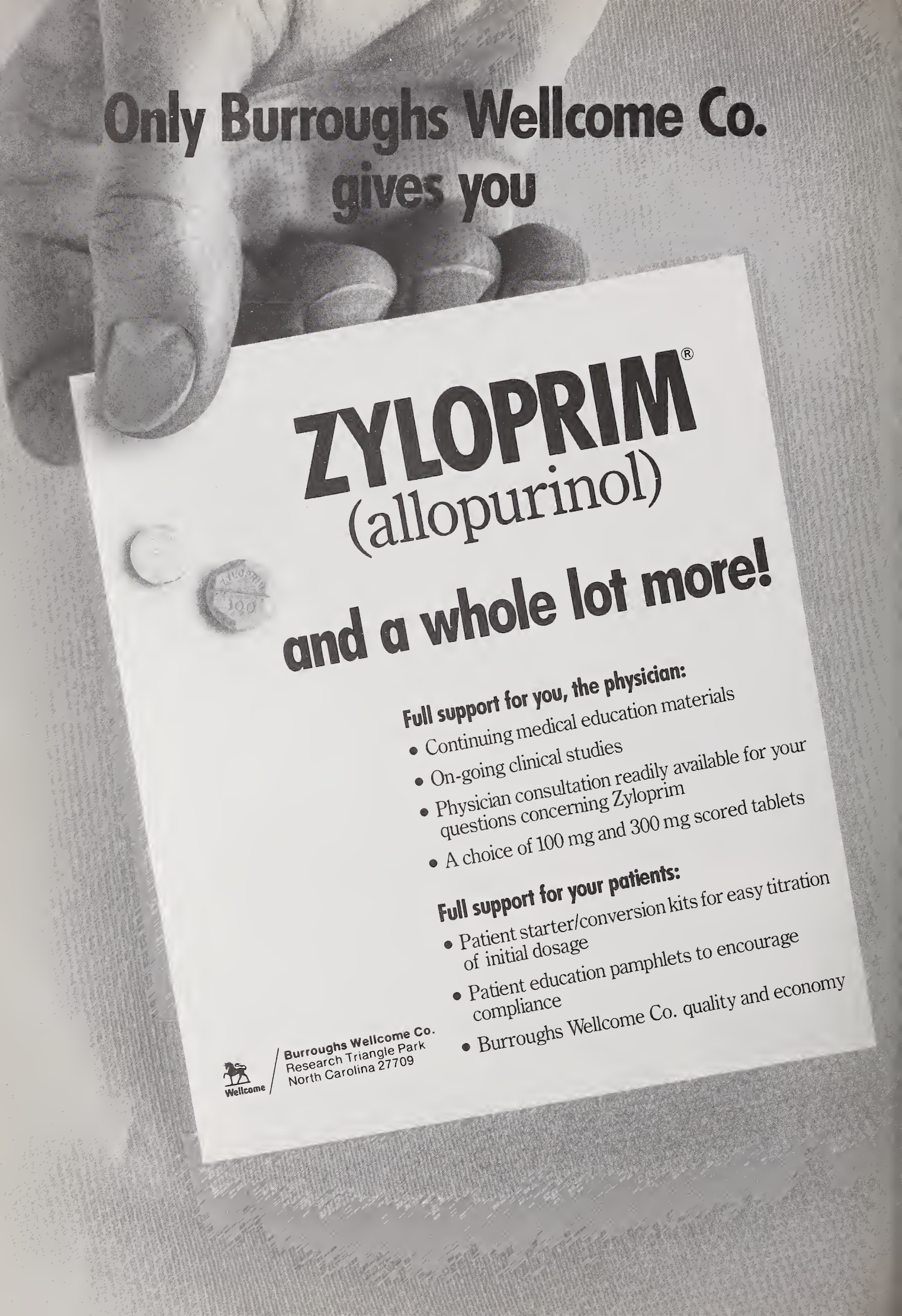
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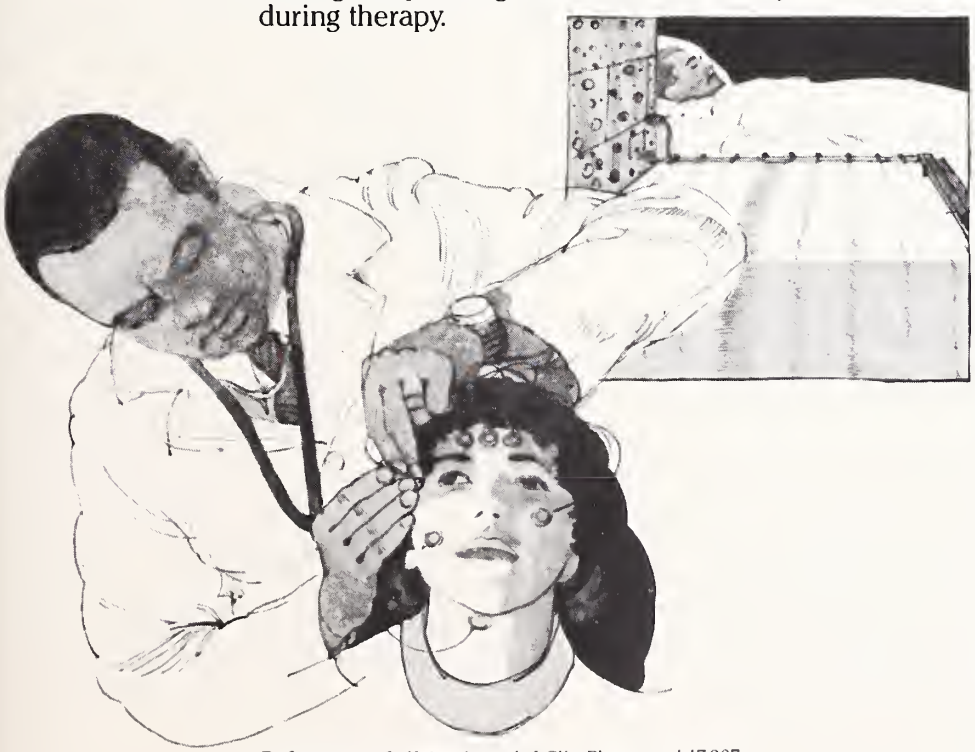
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- During long-term therapy, which is seldom required, periodic blood, kidney and liver function tests should be performed.
- Contraindicated in patients who are pregnant or hypersensitive to flurazepam.
- Caution patients about drinking alcohol, driving or operating hazardous machinery during therapy.



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Dalmane® Ⓥ
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Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

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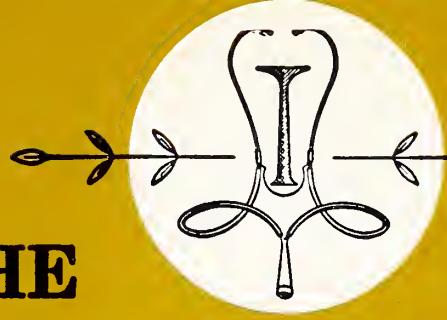
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DECEMBER
1983

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The JOURNAL of the KANSAS MEDICAL SOCIETY

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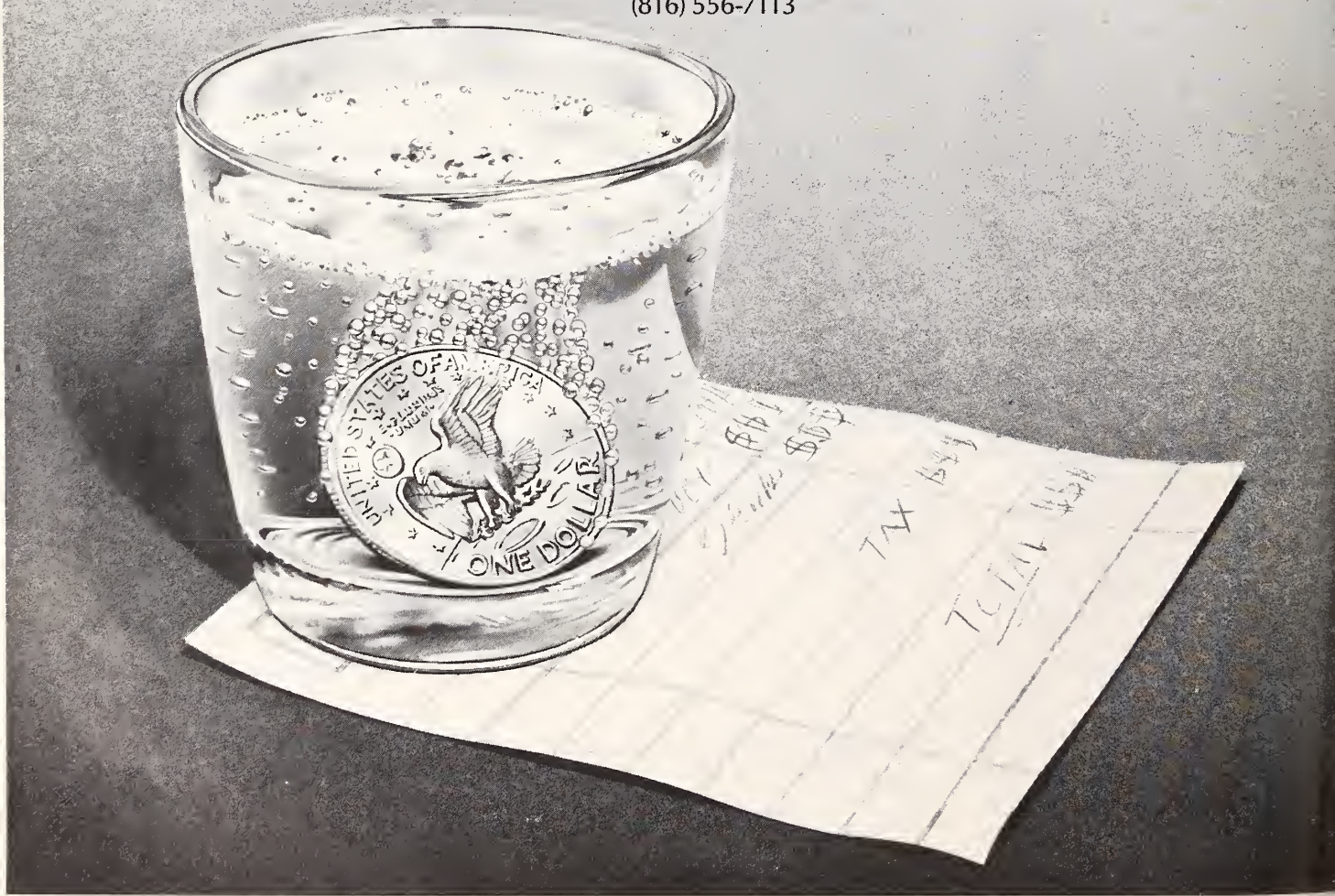
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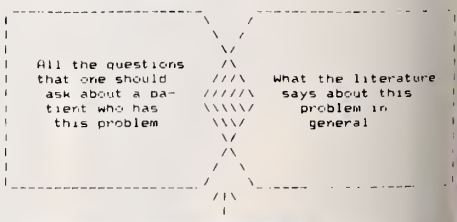
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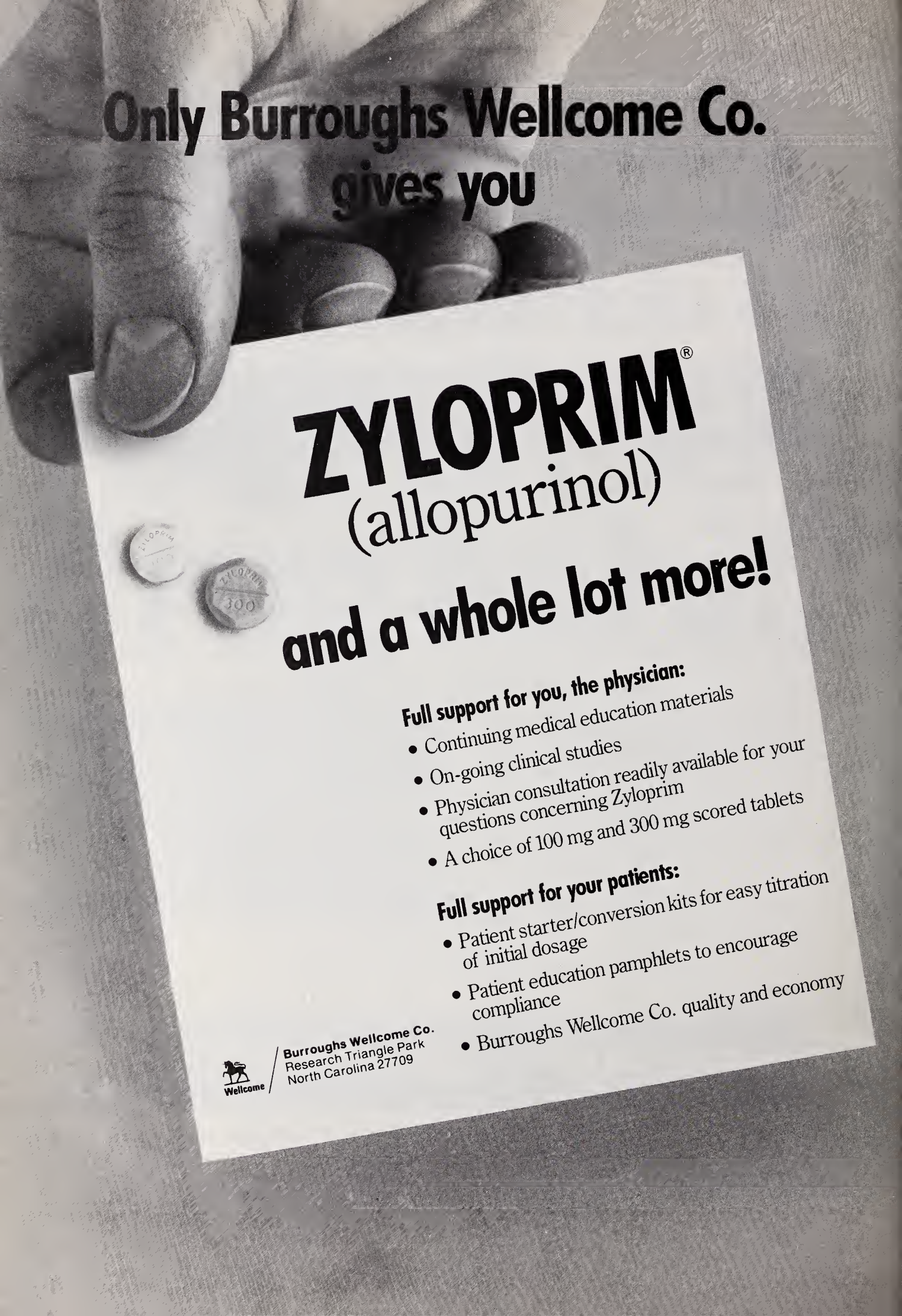
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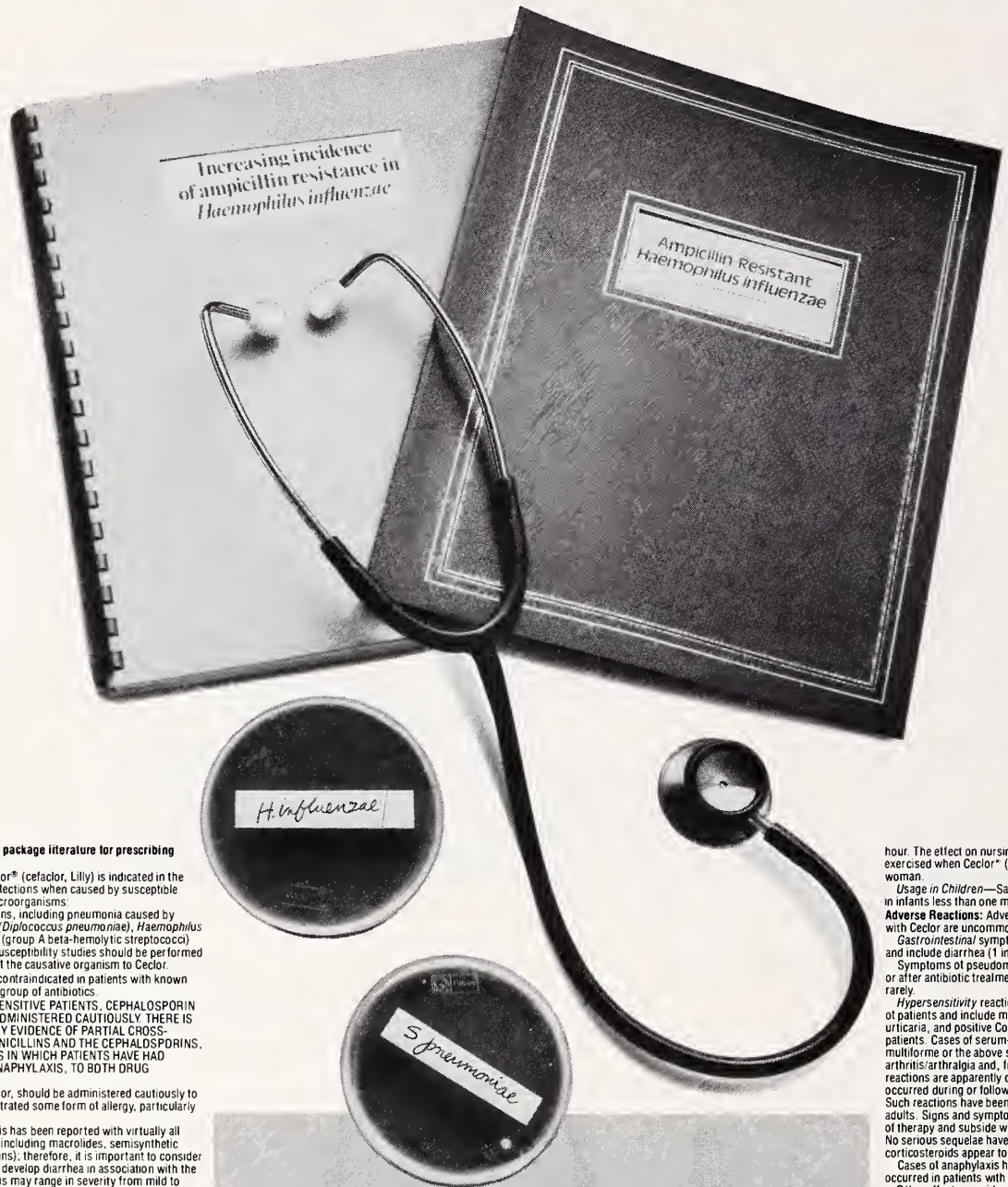
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Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out.

Precautions: *General Precautions*—If an allergic reaction to Ceclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers—Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor* (cefaclor, Lilly) is administered to a nursing woman.

Usage in Children—Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below:

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations of SGDT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

[061782R]

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.[5]
Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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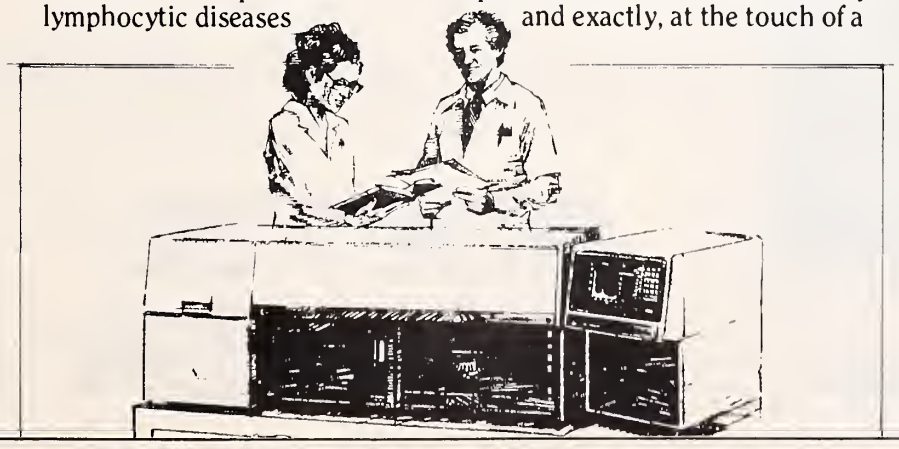
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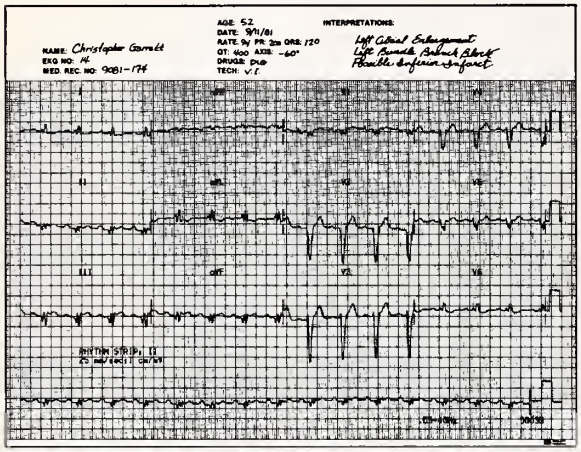


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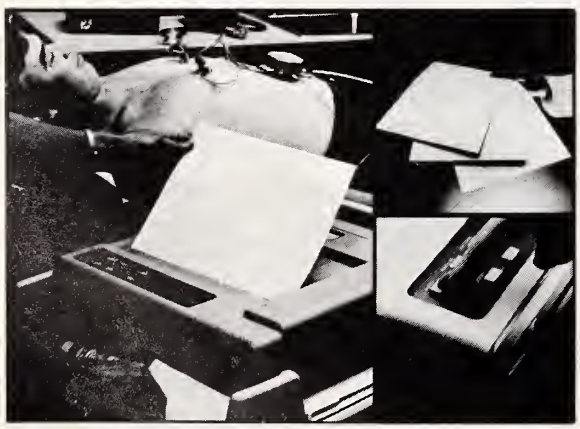


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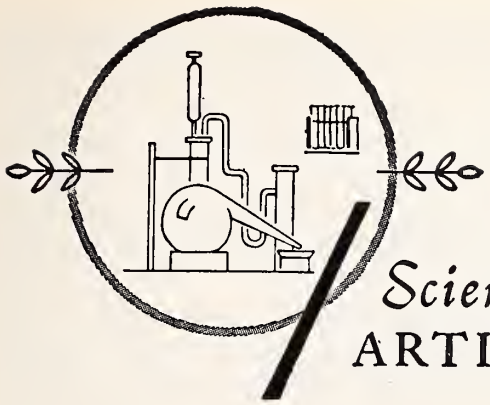
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Intranasal Sphenoethmoidectomy

A Safe and Effective Procedure

PHILIP D. KANNEL, D.O., *Kansas City, Kansas*

Controversy surrounding sphenoethmoidectomy, as stated by Friedman,[1] has centered on three questions: Is major surgery appropriate in treating allergy, a medical problem? If so, should it be performed via the intranasal route? Should the middle turbinate be resected or preserved? Sampter and Lederer[2] believed that nasal polyps appearing later in life should be left alone as their removal might precipitate the appearance of bronchial asthma. This has since been refuted by Schenck,[3] Brown, Harner and Van Dellen,[4] and others.[5] They have concluded that "nasal polypectomy is safe in patients with asthma and intolerance to aspirin."[4] Asthma may develop in the allergic patient following nasal surgery. The nose is the primary entrance for inhalant allergens and is a "shock organ" where allergic reactions mediated by IgE antibodies occur in sensitized mucosa. Surgery does nothing to diminish or change IgE; therefore, the surgeon should not consider allergy a contraindication. The task is purely a mechanical one: to relieve obstruction, to resect nonphysiologic and diseased tissue, and to provide a nasal airway with drainage for all nasal and sinus compartments. The underlying allergies must still be treated, and in most cases are more easily managed without the added burden of repeated sinusitis and nasal obstruction.

Harris Peyton Mosher[6] is generally credited by most English authors with the initial description, in 1912, of the technique of intranasal ethmoidectomy. With this procedure, he advocated complete removal of the middle turbinate. Skillern,[7] a renowned rhinologist, at the same time strongly supported this technique. Pratt[8] and H. L. Williams[9] were more conservative in their approach to intranasal ethmoidectomy and advocated the preservation of the middle turbinate. Inadequate surgery for hyperplastic rhinosinusitis is probably more harmful than no surgery at all, and is to be avoided. Davison,[10] by 1969, had become convinced that extensive disease required extensive surgery, and he advocated his "intranasal pansinus operation." This included, in advanced cases, a submucous resection, removal of polyps, linear cautery of inferior turbinates, excision of the lateral half of each middle turbinate, cauterization of septal hypertrophies, bilateral intranasal ethmoidectomy, and bilateral antrostomies.

Advantages and Disadvantages

The advantages of intranasal sphenoethmoidectomy are: There is no external scar; blood loss with this procedure is usually less than is experienced with external ethmoidectomy; and nasal packing is usually not required unless septal surgery has also been performed. The disadvantages are: Difficulty in teaching the procedure to residents and other otolaryngologists; a somewhat less direct visualization of the structures than would be obtained with the external approach; and a greater risk of injury to vital structures. Mosher[11] stated that the intranasal

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ethmoidectomy operation is technically easy to perform, but in practice can be an easy way to kill a patient.

Indications

Indications for intranasal ethmoidectomy, according to Freedman and Kern,[12] are: Obstructive nasal polyps; chronically infected ethmoid mucosa; and obstruction of maxillary and frontal ostea and ducts in the region of the middle meatus. Taylor *et al.*[13] expanded these indications to include "ethmoid" headaches and acute ethmoiditis that was complicated and unresponsive to conventional therapy with antibiotics.

Surgical Technique

This surgery can best be performed under local anesthesia. It is done with the patient in a semi-sitting position. The nasal passages are anesthesized using a cocaine spray or packing. A sphenopalatine bloc, using 2% xylocaine with 1:100,000 epinephrine, is followed by anterior ethmoidal infiltration accomplished by injecting directly into the middle turbinate and lateral nasal walls. A septoplasty or submucuous resection is done if necessary for visualization, and the surgeon then does an initial polypectomy and resects the middle turbinate, leaving approximately .5 cm attached at its superior portion. With prior knowledge from the sinus x-rays as to the size and location of the sphenoid sinus, the surgeon resects the posterior ethmoids and cannulates the sphenoid sinus. The anterior wall of the sphenoid sinus is completely removed, and the lateral wall and roof of the sphenoid are used for guidelines. In this way, vital structures in the area are exempted from harm. Dissection is then continued from posterior to anterior. All dissection is also kept lateral to the middle turbinate attachment to avoid the cribriform plate. Again, the optic nerve, carotid canal, cribriform plate, lamina papyracea, fovea ethmoidalis, and cavernous sinus are not at risk so long as forceps are never used outside the anterior extention of the planes of the roof and lateral sphenoid wall. The nasal frontal duct is not cannulated. Intranasal antrostomies are performed on each side. There is usually very little bleeding associated with this, and packing is not required unless septal surgery has been performed.

Results

Results have been encouraging. Friedman[1] reported a recurrence of hyperplastic disease following intranasal sphenoethmoidectomy at approximately 31 per cent. Taylor, Crocker, and Keebler[13] had a "cure rate" of approximately 70 per cent which compares favorably to Friedman's rate. Eichel,[5] in a 12-year study, had an overall success rate in controlling nasal polyposis of 83 per cent. This procedure is also felt to benefit patients with asthma and aspirin intolerance and, according to Friedman,[4] reduced the steroid requirement in these patients. In this series of 14 patients, the steroid requirement was reduced in seven and completely eliminated in five, with two remaining unchanged. Aspirin sensitivity of the patient with aspirin triad probably does not have any additional prognostic implication for intranasal surgical treatment other than its association with the more severely asthmatic patients. These patients, however, can benefit greatly from intranasal surgical treatment providing the surgery completely eliminates all hyperplastic disease and provides adequate drainage for all sinuses.

Complications

When the surgery is done by experienced intranasal surgeons, the complications are acceptable and usually not serious. Taylor, Crocker, and Keebler[13] reported on 526 intranasal ethmoidectomies performed on 285 patients with a 2.5 per cent complication rate which included no blindness, meningitis, or death. Freedman and Kern have reported on 1,000 intranasal ethmoidectomies with a 2.8 per cent complication rate which again included no death or blindness. Friedman[1] reported a complication rate of 5.9 per cent for 68 patients, none of which resulted in permanent sequellae. Eichel[5] reported on 236 procedures performed on 123 patients during a 12-year period with a 1.7 per cent major complication rate; these complications included one CSF leak and one pneumoencephalos-meningitis. It has been reported that many patients undergo acute asthma attacks upon the removal of nasal polyps; however, this can be prevented or at least attenuated by use of preoperative steroids and bronchodilators. This can also be reversed in the operating room with intraoperative infusion of steroids and bronchodilators.[4, 14]

Summary

Intranasal sphenoethmoidectomy can be a viable procedure for patients with chronic hyperplastic rhinosinusitis, and for patients with aspirin triad. This surgical procedure is advocated for patients with chronic hyperplastic rhinosinusitis and is a relatively safe procedure when performed by an experienced intranasal surgeon. Also, successful completion of this surgery may permit patients with aspirin triad to undergo easier medical management of asthma.

References are available from Dr. Kannel, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.

Cardiac Pacemakers

Recent Advances

J. E. BOSILJEVAC, JR. M.D., *Emporia*

PERMANENT CARDIAC pacemaker technology has advanced considerably since the advent of the first implantable unit in 1959. Most recently, the size of the pulse generator and its longevity have been improved. On the immediate horizon is the development of dual chamber pacemakers, which pace and sense in both the atrium and the ventricle.

There have also been improvements in the technique of placement. This includes the type of leads available for placement, as well as such methods as the use of the catheter introducer. Finally, pacemaker followup, the ability to reprogram various pacemaker parameters, and the measurement of chronic thresholds have increased the safety of cardiac pacemakers.

Technique

The technique used for placement of permanent pacemakers was described in an earlier paper.[1] The data presented here concern pacemakers implanted during an 18 month period. A total of 28 patients underwent pacemaker implantation, 17 females and 11 males. The average age of the group was 77 years. *Table I* indicates the reason for pacemaker placement, comparing this with data from an earlier study. Sinus node dysfunction includes sinus bradycardia with syncope and the tachycardia-bradycardia (sick sinus) syndrome. Patients with atrial fibrillation and slow ventricular rate were symptomatic with syncope or congestive heart failure.

The catheter introducer was used in nine patients (30%), and consists of a needle and guide wire, over which a large sheath may be placed into the subclavian vein using the Seldinger technique. The endocardial lead is then threaded through this sheath. The catheter introducer enables cannulation of the subclavian vein for placement of the endocardial lead if the cephalic vein in the deltopectoral groove is inadequate. The advantage is that this can be used through the initial operative incision. If necessary, two catheterizations using the introducer may be performed on the same side to place an atrial and a ventricular lead for dual chamber pacing.

The long tine tip unipolar endocardial lead was used in each case (Medtronic 6972). Endocardial lead displacement occurred in one patient. This was resolved at initial repositioning of the endocardial lead. No other complications occurred, and there was no mortality in this series.

Discussion

Transvenous placement of permanent cardiac pacemakers continues to be a safe procedure.[1-3] This is further supported by the absence of mortality and the low complication rate in the present study. Probably the biggest factor contributing to this safety is the use of local anesthesia for implantation of permanent pacemakers, since these patients are usually elderly with heart disease and other medical problems.

Improvements in the available endocardial leads have also contributed to this safety. The present endocardial leads are smaller in diameter. Their use decreases the possibility of cardiac perforation and tamponade, which is the most serious complication that may occur with implantation of permanent pacemakers. Another advantage of the lead used in this series is that the long tines become entwined in the endocardial trabaculae *(Figure 1)*. This provides

TABLE I
DIAGNOSIS REQUIRING PERMANENT PACEMAKER

	Original Study[1]	*Present Series*
Complete heart block	205 (47%)	9 (32%)
Sinus node dysfunction	178 (41%)	10 (36%)
Atrial fibrillation with slow ventricular response	27 (6%)	9 (32%)
Post acute myocardial infaretion	15 (3%)	
Drug resistant ventricular arrhythmias	10 (2%)	
Congenital heart block	3 (1%)	
	438	28

Presented at the 34th Annual Meeting of the American Heart Association, Kansas Affiliate, July 8, 1983, in Kansas City, Missouri.

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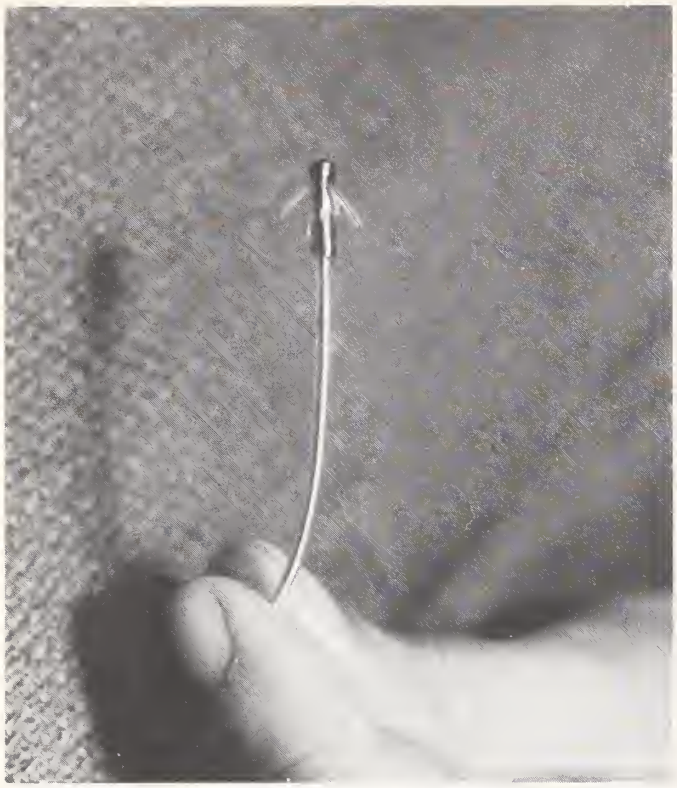


Figure 1. Tine tip electrode — the tines provide fixation to the endocardial trabeculae.



Figure 2. Comparison of sizes of old and present pacemakers.

better fixation, decreasing the possibility of lead displacement. The displacement rate in the present series was 3 per cent. In the prior series[1] there was a 10 per cent displacement rate, where a flange or smooth tipped electrode was used in most patients.

The availability of the catheter introducer has also simplified placement. With this instrument, the original operative incision may be used even if there is no available cephalic vein in the deltopectoral groove. If dual chamber pacemakers are used, the small diameter of the endocardial leads allows placement of both leads in the cephalic vein. Otherwise, the catheter introducer technique allows placement of two leads through the subclavian vein.

The current pulse generators (Medtronic-Spectrax SX 6985) are smaller and more comfortable to the patient *(Figure 2)*. They weigh less than 2 ounces and have an anteroposterior diameter of 6 millimeters. A lithium-iodide power cell is used as the energy source. With an excellent electrode position and low stimulating thresholds at initial placement, the battery life of the present pulse generators should be in the range of eight to ten years. Nuclear powered pacemakers are still in the experimental stage and not widely used.

Postoperative followup has been facilitated using a telephone monitoring device. Patients who live some distance from their physician may simply call in and, using this device, an EKG may be recorded in the physician's office. This EKG can be used to confirm proper pacemaker function and indicate any early signs of possible pacemaker failure.

Also, various parameters of the current pulse generators can be reprogrammed. The rate of the pacemaker may be adjusted according to the patient's needs. In conjunction with drug therapy, this may help control some tachyarrhythmias and also premature ventricular contractions. Chronic threshold measurements determine the energy required to pace the heart. The energy output from the pulse generator is proportional to the pulse width and amplitude of the electrical impulse. If measurements three months after implantation demonstrate that the patient does not require the standard pulse width and amplitude settings for cardiac pacing, adjustments can be made. Decreasing the pulse width or amplitude in some patients should increase the pacemaker battery life.

On the horizon is the advent of dual chamber pacing,[4] which is called physiologic pacing. One problem seen with ventricular pacing is the so-called "pacemaker syndrome." Retrograde atrioventricular conduction of the pacing impulse may cause hypotension, arrhythmias, dyspnea, and even heart failure. Some patients with sinus bradycardia and poorly controlled premature ventricular contractions or other arrhythmias may also benefit from physiologic pacing.

Synchronizing atrial and ventricular contractions increases cardiac output up to 25 per cent by provid-

(Continued on page 623)

Biliary Cysts and Carcinoma

Implications of Significant Incidence of Association

TERENCE WADE, M.D.; LARRY J. BUTLER, M.D. *and*
RICHARD I. MacARTHUR, M.D., *Kansas City, Kansas*

CYSTIC DILATIONS of the biliary tree occur in several forms, most frequently involving the common bile duct. The first reported case dates to 1723, but the first well-documented case was presented by Douglas in 1852.[1] The disease is relatively common in the Orient, although rare in the United States, and has been most extensively reviewed by Powell[2] in the English, and by Yamaguchi[3] in the Japanese literatures. Biliary carcinoma associated with biliary cysts is exceedingly rare, with only 65 cases reported in the world literature,[4, 5] most from Japan.

The following discussion of the major aspects of biliary cystic disease, operative treatment, and its implications regarding development of carcinoma in the cyst is presented with a report of the seventh American case of cyst-associated biliary carcinoma.

Epidemiology

Bile duct cysts were first classified into three types in Alonso-Lej's 1959 review,[6] with Type I as common bile duct cyst, Type II as common bile duct diverticulum, and Type III as dilation of the intraduodenal bile duct. Caroli[7] later described combined intrahepatic and extrahepatic cystic disease, and this has been called Type IV *(Figure 1)*.

Type I cysts are the most common, representing the majority of all types. The frequency of cyst-type from various recent reviews is summarized in *Table I*.

Biliary atresia has been noted in association with biliary cysts in neonates. Flanigan[8] noted that 2 per cent of the cases of biliary cysts he reviewed had associated biliary atresia. Five of the 11 infants and children reported recently by Lilly[9] had histiopathologic findings consistent with biliary atresia. In Kim's[10] survey of the surgical section of the American Academy of Pediatrics, 24 of 178 cases reported had biliary atresia. Kim considered choledochal cysts with biliary atresia to represent an operable form of extrahepatic bile duct atresia, and notes that 11 of 22 patients followed are living without liver disease.

The age at diagnosis of patients with choledochal cyst is quite variable, but the majority are diagnosed in early life. Flanigan[8] reported 25 per cent of cases diagnosed before one year of age and 60 per cent before age ten years. However, he also noted that 23 per cent were diagnosed in patients older than 20 years. The age distribution is similar in the Japanese review by Yamaguchi.[3] There is a marked female predominance for biliary cysts. Flanigan and Yamaguchi noted 81 and 75 per cent females in their respective studies.

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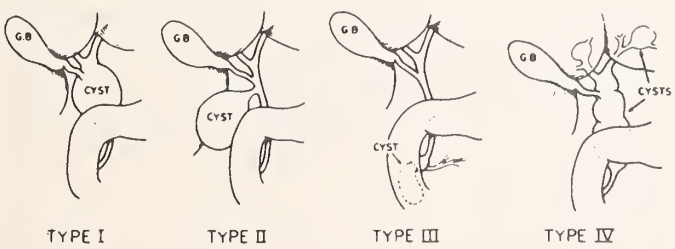


Figure 1. Anatomic classification of biliary cystic disease.[2]

TABLE I
FREQUENCY OF BILE DUCT CYST TYPES

	Flanigan[9]	*Yamaguchi*[3]	*Kim*[9]	*Powell*[2]
Number of Cases	760	878	188	1076
Type I	86.7%	77.7%	93.1%	84.8%
Type II	3.1%	2.0%	5.3%	2.8%
Type III	5.6%	1.4%	1.6%	5.1%
Type IV	2.6%	18.9%	NA	7.3%
Associated biliary atresia	2.0%	NA	12.8%	NA

Pathology and Etiology

The histopathology of the different types of cysts is very similar. There is a wall of variable thickness that is made up of dense collagenous tissue and smooth muscle, with some degree of inflammatory infiltrate usually present. The mucosa lining is often absent or present in a patchy distribution made up of cylindrical or columnar cells. Type III cysts are usually lined with duodenal type mucosa.

The etiology of bile duct cysts is largely unknown. The main controversy concerns congenital *vs* acquired origin of cysts. It is generally accepted that cysts may be congenital in origin, and this is supported by finding of bile duct cysts in neonates. However, a number of cases suggesting acquired lesions have been reported.

Presentation and Diagnosis

The classic triad of symptoms of bile duct cyst is abdominal pain, jaundice, and abdominal mass. Unfortunately, this combination is seen in only 13-38 per cent of patients. Jaundice is present in more than one-half of patients in the larger series, with pain and abdominal mass seen in decreasing frequency.

The duration of symptoms is quite variable, but 81 per cent of patients reviewed by Flanigan had symptoms for more than one month, and 48 per cent had at least a six-month history of symptoms. Histories of intermittent right upper quadrant pain and jaundice for several years in elderly patients are not uncommon.

The diagnosis of biliary cysts was rarely made preoperatively in the past, but new diagnostic studies and increased suspicion have made the diagnosis more common. Preoperative diagnosis is still the exception rather than the rule with reported rates of 27-85 per cent. The diagnosis should be considered in patients who present with intermittent abdominal pain and jaundice or right upper quadrant mass. Standard contrast roentgenography may be useful if upper gastrointestinal studies demonstrate displacement of the antrum or duodenal bulb. Similar organ displacement may be noted on barium enema or intravenous cholecystography. Technetium radionuclide scintiscans have been of some use. Ultrasonography is good for cyst location and size but will not prove biliary origin, and computerized axial tomograms have been used. If a cyst malignancy is suspected, angiography is important to guide the operative approach.

Definitive diagnosis is based on visualization of the biliary tract by intravenous cholangiography (IVC), percutaneous transhepatic cholangiography (PTHC), or endoscopic retrograde cholangiopancreatography (ERCP). ERCP is not a benign procedure and should probably be reserved for cases in which IVC and PTHC fail to visualize the tract. Operative cholangiography will occasionally demonstrate a previously unsuspected cyst.

Associated Complications

Associated stones have been reported in two to eight per cent of the cases of biliary cysts.[3, 8] The stones are mostly gallstones located in the cysts, but extracyst gallstones and pancreaticolithiasis have also been noted. Pregnancy has been associated with biliary cysts and 23 cases of cyst rupture during pregnancy have been reported.[8] Bile peritonitis resulting from traumatic or spontaneous rupture has also been reported in 16 cases. Other less common complications include portal vein thrombosis, massive gastrointestinal bleeding, pancreatitis, and pancreatic cyst.

Treatment

Treatment for biliary cysts is unquestionably surgical. Tsardakas'[11] early study showed that 29 of 30 patients treated medically died from their disease. The best procedure for treatment has been a matter of controversy, but increasing data supporting cyst excision have been presented in the last few years.

In the past, internal cyst drainage procedures such as Roux-en-Y choledochocystojejunostomy (RYCCJ), choledochocystoduodenostomy (CCD), and choledochocystojejunostomy (CCJ) were the mainstays of treatment in this country, with RYCCJ being advocated as the treatment of choice for Type I cysts. Early reports of mortality as high as 15 per cent for cyst excision contributed to the preference for the alternative procedures. However, newer studies have shown decreased mortality with the excision procedure, and Flanigan reported no significant difference in operative mortality between RYCCJ and excision in cases reported between 1968 and 1972. He did note a very significant difference in morbidity, however. A morbidity rate of 34 per cent for RYCCJ was found as compared to a rate of 8 per cent for excision, and while none of the patients treated with excision required reoperation, 13 per cent of those undergoing RYCCJ required a second procedure. Powell's[2] data from cases from 1975-1980 confirm Flanigan's findings and are shown in *Table II*.

The associated morbidity seen with internal drainage procedures is related to several factors. Ascending cholangitis, anastomotic stenosis with recurrent jaundice, pancreatitis, cholelithiasis, and carcinoma all have been late complications of procedures leav-

TABLE II
POWELL[2] 1975-1980

Treatment (Number)	*Compli-cations*	*Mortality*	*Reoper-ations*
Primary excision (61)	1.6%	1.6%	1.0%
Secondary excision (22)	4.5%	4.5%	0
All internal drainage (91)	10.0%	12.0%	22.0%
CCD (38)	18.0%	16.0%	17.0%
CCJ (14)	7.0%	14.0%	29.0%
RYCCJ (39)	2.0%	8.0%	5.0%

ing the cyst behind. Recurrent cholangitis is the most common and often leads to reoperation, as can stenosis of the anastomotic site. The formation of carcinoma in the cyst left behind has become an important concern and Todani[4] notes that in the 63 cases of biliary cyst carcinoma he reviewed, 36 (57.1%) of the patients had undergone previous internal drainage procedures.

Excision of the biliary cyst eliminates most of the potential problems seen in internal drainage procedures, and has long been the advocated procedure in Japan. Excision eliminates any reservoir for bile stasis that can be left with internal drainage procedures. This reduces the likelihood of chronic infection and stone formation. With excision, the possibility of pancreatic reflux into a retained cyst and the resulting cholangitis are also eliminated. Finally, cyst excision probably eliminates the possibility of bile duct carcinoma by removing a mucosa that is chronically inflamed and has a high carcinogenic potential.

Recent reports in the United States and Canada have shown very good results with cyst excision. Lilly[12] reports successful cyst excision without mortality in 11 consecutive infants and children. Fitler[13] reported seven cases without mortality and with prolonged drainage from peritoneal drains in two patients as the only complications. With this type of experience and the recommendation of excision by reviewers like Flanigan, Powell and Yamaguchi, cyst excision will probably become the treatment of choice for Type I cysts.

Various techniques have been described for cyst excision but most have been unpopular due to difficulty in separating the thick, often inflamed, cyst wall from the underlying adherent structures of the porta hepatis. Recently Lilly has described a technique that confines the excision to the inside of the cyst on its posterior wall. This procedure offers the advantage of avoidance of damage to the vascular, biliary, and pancreatic structures underlying the posterior wall, and has been noted to be helpful especially in cases involving inflammation.

Reconstruction after cyst excision has been accomplished in many ways. Roux-en-Y hepaticojejunostomy (RYHCH), Roux-en-Y choledochojejunostomy (RYCDJ), or hepaticoduodenostomy (HCD) have been the most widely used, but there are cases in which hepaticocholedochostomy was possible after cyst excision. Hepaticoduodenostomy with a wide stoma is recommended by Todani,[4] but apprehension about reflux of duodenal contents into the biliary system have made RYHCJ or RYCDJ the most common procedures in the United States. Most authors agree that any procedure should include cholecystectomy.

Treatment of the other less frequent types of biliary cysts has also been outlined in the literature. Type II cysts are usually easily treated with excision of the cyst after ligation of the diverticular stalk. Type III or choledochoceles are treated by transduodenal resection with choledochoduodenostomy. Type IV disease with multiple cysts does not allow one specific recommendation, and the procedure used must also be dictated by the individual anatomy, but extrahepatic cysts can often be managed in the recommended way with no accompanying treatment for intrahepatic cysts.

Carcinoma

The most dreaded complication of biliary cyst is associated carcinoma. Flanigan[8] noted a rate of about 2.5 per cent, but other reviewers have suggested an incidence as high as 15 per cent in patients with biliary cysts. This is compared to an incidence of 0.0007-0.1400 per cent of hospital admissions with biliary carcinoma. The female-to-male ratio is 2-2.5:1, and two-thirds of the patients were less than 30 years of age,[4] the youngest being five years old.[14] Adenocarcinoma is the most common cell type, but squamous cell, anaplastic, undifferentiated, and adenoacanthoma types have also been noted. Twenty to twenty-five per cent of the reported patients had associated gallstones. This prominent association of biliary cyst and biliary carcinoma is an important argument for excision, rather than bypass, of cysts. Also, since the carcinoma arises in the mucosa, the mucosal-stripping procedure described by Lilly[12] allows removal of the potential source of cancer while sparing the portal structures.

(Continued on page 623)

Lead Poisoning

Children Who Have Been Treated With Folk Remedies

Mexican Folk Remedies — California

In May 1982, a 15-month-old California child and his 3-year-old sibling were treated in Mexico with multiple doses of azarcon (lead tetroxide) for chronic diarrhea that had been unsuccessfully treated with ampicillin. In June, the children were taken to a San Diego hospital where the younger child was found to have a blood lead level measurement of 124 μg/dl; the 3-year-old expired with seizures. It was not known whether an autopsy was performed, but azarcon-induced lead encephalopathy was suspected as a cause of death.

Because of these cases, in June 1982, the Los Angeles County Department of Health Services surveyed residents of six predominantly Hispanic, geographically representative census tracts in an attempt to estimate exposure to and knowledge of azarcon and greta (lead oxide). A total of 545 systematically selected households were included. Familiarity with the substances was greatest among Mexican-Hispanics, and prior use was exclusive to this group. Respondents in approximately one-quarter of Mexican-Hispanic households were familiar with one or both of the substances by means other than media announcements. An estimated 7.2%-12.1% of Mexican-Hispanic families admitted prior use from "years ago" to within the past month. One respondent provided interviewers with azarcon from the family medicine cabinet. Since investigators noted a reluctance to admit using azarcon or greta, the incidence of ingestion might have been greater than results of the survey indicated.

A Colorado survey in June-September 1982 among Texas farm workers showed that 7.0% of 100 migrant children under 12 years of age had been treated with substances called azarcon or greta at some time for gastrointestinal illness. Other states with migrant populations, such as Arizona, New Mexico, and Texas, are presently investigating the problem.

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Editorial Note: In summer 1981, the first cases of lead poisoning associated with the Mexican folk remedy, azarcon, were identified in Los Angeles, California, and Colorado.[1, 2] Since that time, nine additional confirmed cases associated with the ingestion of azarcon or the related remedy, greta, have been reported in California. In addition, five cases have been reported from Michigan and Wisconsin.

Greta and azarcon are fine powders with total lead contents varying from 70% to greater than 90%. As powder, they provide a large surface area for potential absorption. These remedies apparently are most often administered to infants and children, who are the most susceptible in terms of clinical impact and the capacity to absorb lead.

With the identification of multiple cases of lead poisoning and indication of significant exposure, major media efforts publicizing the dangers of azarcon and greta have been directed at Hispanic communities in California. Until recently, these substances were available at herb shops and from folk healers on both sides of the Mexican-American border. The U.S. Food and Drug Administration (FDA) has initiated a national recall of greta, and a reported 25 pounds of the substance was recently seized in southern Texas. The FDA is currently investigating the sale of greta in the Yakima Valley of Washington

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State. Mexican health authorities have reportedly instituted recall efforts in Baja California, Mexico.

Health professionals are urged to report cases of azarcon- or greta-associated lead poisonings and to promote educational programs in their Hispanic communities regarding the dangers of these folk remedies. Health education material in Spanish and English is available from the State of California and Los Angeles County Departments of Health Services and from the Sunrise Community Health Center, P.O. Box 245, Greeley, Colorado 80632.

References

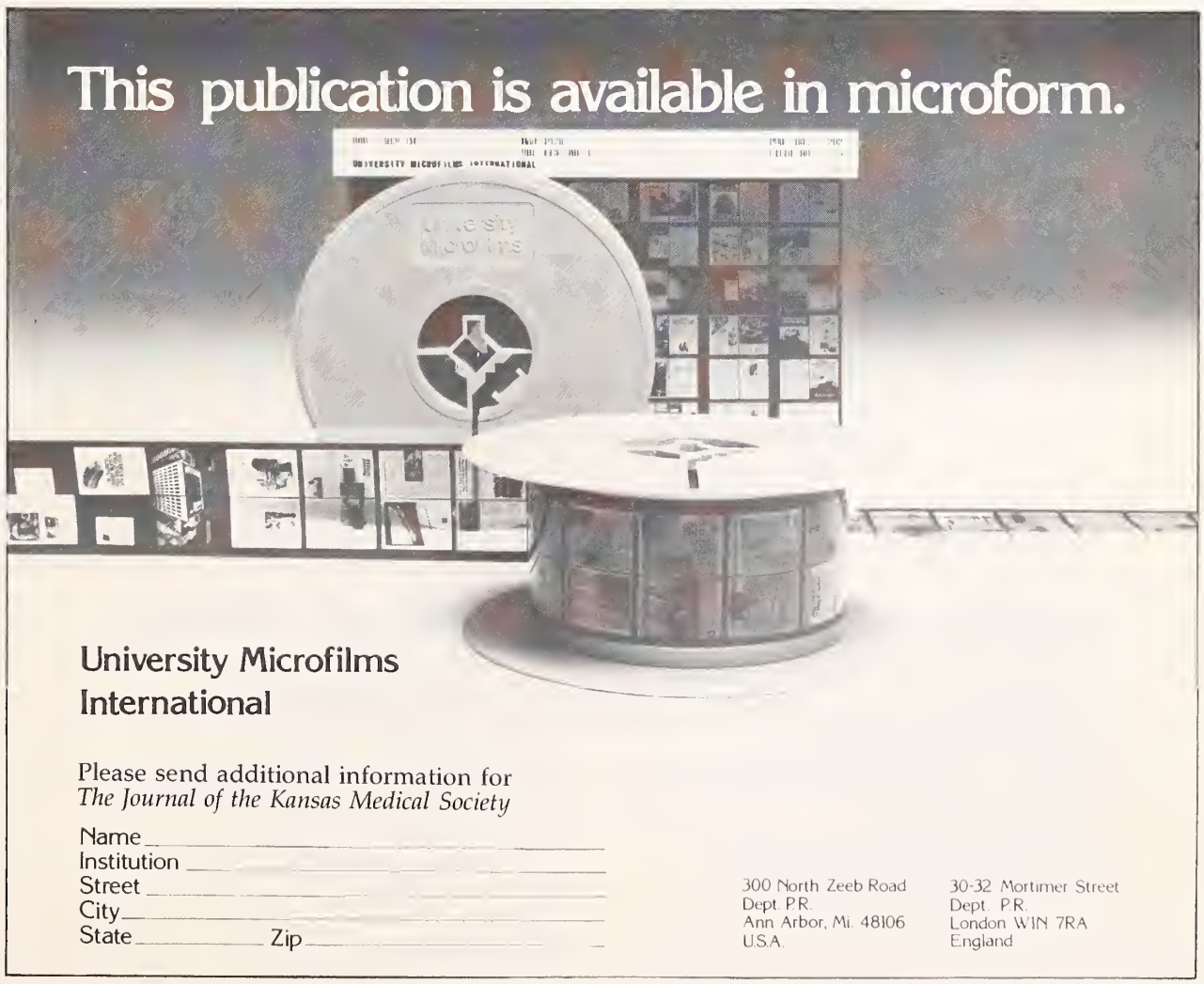
1. CDC. Use of lead tetroxide as a folk remedy for gastrointestinal illness. MMWR 1981;30:546-7.
2. CDC. Lead poisoning from lead tetroxide used as a folk remedy — Colorado. MMWR 1981;30:647-8.

Hmong Children — Minnesota

Between January 1, and June 30, 1983, 35 children with lead toxicity* were identified through routine screening by the St. Paul, Minnesota, Division of Public Health. Of these, 24 (69%) were Hmong refugees from Northern Laos. This represents a twofold to threefold increase in the number of Hmong children found to have lead toxicity in St. Paul compared with previous years. One source of lead poisoning appears to be a Hmong folk remedy used for treating infants and children with fevers. A case report follows:

On May 3, 1983, a 6-month-old Hmong girl was found to have lead poisoning (blood lead [BL] 60 μg/dl, erythrocyte protoporphyrin [EP] 263 μg/dl, hematocrit 38%) during screening for well-baby care. She was asymptomatic at the time. Her physical examination was unremarkable. X-ray films of the wrists and knees revealed dense provisional zones of calcification suggestive of lead deposits. No environmental sources of lead, such as paint, could be identified after a thorough investigation of the family's home. After detailed questioning by the

* Blood lead ≥ 30 μg/dl and erythrocyte protoporphyrin (EP) ≥ 60 μg/dl, determined by hematofluorometer. The St. Paul Division of Public Health considers EP of 60 μg/dl elevated instead of the CDC recommendation of 50 μg/dl because of the differences in calibration of their hematofluorometer.



child's pediatrician, the parents admitted giving red and orange powders to the baby as a cure for high fever. Laboratory analysis of the red powder showed a lead concentration of 8%. The infant was given ethylene diamine tetraacetic acid (EDTA) chelation therapy as an outpatient.

Officials have been unable to obtain samples of the folk remedy from the parents of other Hmong children with lead poisoning. The remedy, generally referred to as "pay-loo-ah," consists of red and orange powders, the composition and source of which often vary; therefore, a more exact description of the material remains difficult. Believed to have originated in China or Southeast Asia, pay-loo-ah is fed to children as a cure for fever or rash. Samples of folk remedies were obtained from several Hmong households in the community, and the U.S. Food and Drug Administration confirmed that two contained lead (1% and 90%). Arsenic was found in three samples at concentrations of 70%-80%. These folk remedies were in wide use and were easily available through local Asian food stores or Hmong peddlers. To date, no cases of arsenic poisoning in the Hmong children have been reported.

Reported by C Levitt, MD, Children's Hospital, St. Paul, D Paulson, MD, K Duvall, MPH, J Godes, MPH, St. Paul Div of Public Health, AG Dean, MD, State Epidemiologist, Minnesota State Dept of Health; J Roberts, J Egenberger, Minneapolis District, US Food and Drug Administration; Special Studies Br, Chronic Diseases Div, Center for Environmental Health, CDC.

Editorial Note: Folk remedies have been known to cause lead poisoning. A Mexican folk medicine, azarcon, has been reported to cause lead poisoning in Mexican children in a number of southwestern states.[1, 2] Hmong refugees, who have emigrated from North Laos, have an estimated total population over 50,000 and live in many parts of the United States, with the largest concentrations in Fresno, Stockton and San Diego, California, in addition to St. Paul.

Health-care providers for Hmong and Southeast Asians should be aware of this unusual lead source. Screening for elevated blood lead levels is necessary to identify additional cases, because symptoms of lead toxicity generally have not been reported by the Hmong. Reporting of cases to local or state health departments is recommended. Appropriate health education will be necessary to inform the Hmong of the health consequences associated with this folk remedy.

Lead poisoning resulting from folk remedies exemplifies the need to continue screening young children for lead toxicity. CDC recommends routine screening of EP of all children between 6 months and 5 years of age. An EP level of over the CDC recommendation of 50 μg/dl indicates either iron deficiency anemia or lead toxicity.[3] Lead toxicity should then be confirmed by a blood lead level.

References

1. Bose A, Vashistha K, O'Loughlin BJ. *Azarcón por Empacho* — another cause of lead toxicity. Pediatrics 1983;72:106-8.

2. CDC. Lead poisoning from lead tetroxide used as a folk remedy — Colorado. MMWR 1982;30:647-8.

3. CDC. Preventing lead poisoning in young children: a statement by the Center for Disease Control. April 1978, page 4.

Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.



Drug INFORMATION

JOYCE GENERALI, R.Ph., *Editor*

Cimetidine vs Ranitidine

Question: How does the new histamine H_2-antagonist, ranitidine, compare with cimetidine?

Answer: Since its introduction in 1977, cimetidine (Tagamet; Smith Kline French) has made a major impact in ulcer therapy. In addition, it has become the fifth most prescribed drug in the United States. Until this year it was the only histamine H_2-antagonist on the market.[1] Recently, however, a new H_2-antagonist, ranitidine (Zantac; Glaxo/Roche), was made available in this country. It is indicated for the short term treatment of active duodenal ulcers and pathological hypersecretory conditions such as Zollinger-Ellison syndrome and systemic mastocytosis.

Chemically, ranitidine is unique in this class of drugs because of its alkyl furan ring structure. Cimetidine incorporates an imidazole ring in its structure. Although chemically different, these agents are pharmacologically similar. They exert their antiulcer activity by blocking the histamine H_2 receptor in the gastric mucosal cells thus inhibiting the secretion of acid. The pharmacokinetic parameters of cimetidine and ranitidine are very similar.[3] The biological half-life of each agent is two to three hours. The duration of action for ranitidine, however, is 8-12 hours. Therefore, it can be administered on a twice daily dosage schedule rather than four times a day as with cimetidine.[4]

Ranitidine, in twice daily doses of 100-150 mg, has been shown to be as safe and as effective as cimetidine (200 mg three times a day and 400 mg at bedtime) in short term duodenal ulcer therapy.[5-7] It has not, however, been shown to be any more effective than cimetidine. Ranitidine has also been used successfully to treat some cimetidine resistant Zollinger-Ellison patients.[8]

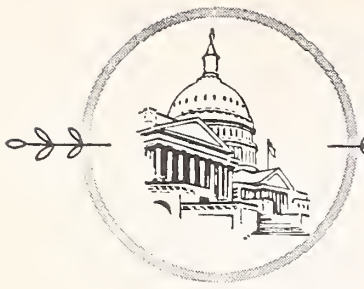
Side effects have been reported in 3-5 per cent of patients treated with ranitidine.[9] The most common effects are headache, skin rash, and dizziness. Ranitidine appears to have little or no antiandrogenic activity.[10, 11] This may indicate a lesser probability of its causing gynecomastia, a side effect associated with long term cimetidine use.[11] To date, clinical use of ranitidine has not been extensive in the United States. It should be noted that additional side effects of cimetidine did not become apparent until long-term clinical experience had been documented.

No drug interactions with ranitidine were reported in the literature. Ranitidine, unlike cimetidine, does not inhibit the mixed function oxidase (P-450) system.[12-14] This is one major cause of cimetidine's interactions with warfarin, benzodiazepines, theophylline, beta-blockers, and anticonvulsants. Both agents potentially can interact with drugs that are affected by the pH of the stomach such as acid labile antibiotics, oral pancreatic enzymes, and aspirin.

In conclusion, early clinical use indicates that ranitidine is an effective agent for treatment of duodenal ulcers and Zollinger-Ellison syndrome. It offers advantages over cimetidine in that it can be administered twice daily (150 mg). In addition, it has little potential for drug interactions. It is presently available in 150 mg tablets. The intravenous dosage form should be available early in 1984.

Submitted by
Howard Montgomery, R.Ph.

References are available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.



Socio-
ECONOMICS

Perinatal Care Centers

Guidelines for Level I Centers in Kansas

Purpose

GUIDELINES for Level I hospitals in Kansas have been formulated by the Kansas Perinatal Care Program to encourage all hospitals with maternity services to achieve the best possible level of care consistent with personnel and geographic considerations. They have been carefully reviewed and discussed, and are felt to represent the minimal requirements by which adequate perinatal care can be delivered in Kansas.

To achieve the necessary level of communication between Level I hospitals and the Kansas Perinatal Care Program, the Kansas Department of Health and Environment has agreed to coordinate inquiries and assist with advising Level I hospitals in their efforts to meet these Guidelines.

Introduction

The Guidelines shall be implemented in accordance with the following:

- Every hospital where babies are delivered should strive for compliance with these Level I Guidelines for obstetrical and newborn care.
- Good obstetrical care is primary to good perinatal care. Obstetricians, pediatricians, and family practice physicians must work together.
- A joint committee is to be established comprised of a physician or physicians, a nurse or nurses, and a member of the hospital administration. The function of this committee is to study perinatal mortality and morbidity within their institution and to designate a representative to attend the quarterly meetings of the state perinatal program.
- Leaders should be identified to carry out liaisions with the community and hospital administration.

Level I, like Level II and III hospitals in Kansas, are those committed to providing the best possible care for pregnant women and babies delivered of those pregnancies. Level I responsibility for this care extends beyond the confines of Level I hospitals. The Level I hospital must have confidence in the referral system and be familiar with it. It must have input into the system and communicate frequently with other levels. Level I hospitals include the small hospital with fewer than 50 deliveries per year as well as the 140 bed hospital with 350 deliveries per year. Their primary mission is to provide care for uncomplicated pregnancies.

Level I hospitals whose staff and facilities are limited must limit their services consistent with their resources. On the other hand, those Level I hospitals with appropriate professional staff and facilities may assume responsibilities for more comprehensive patient care.

Level I Services

It is recommended that Level I Centers provide the following services:

1. Perinatal care for the uncomplicated obstetric patient and normal newborn, as well as for emergencies that could arise. Level I Centers with appropriate professional personnel and facilities may give more comprehensive care.

These guidelines were formulated and recommended by the Kansas Regional Perinatal Care Program Medical Council consisting of the following physicians: John F. Benage, Fort Scott; John W. Calkins, Kansas City; Carl M. Christman, Wichita; John F. Evans, Wichita; Howard A. Fox, Kansas City; Gerald L. Mowry, Manhattan; Russell A. Nelson, Wichita; Patricia T. Schloesser, Topeka; and Donnal C. Walter, Kansas City.

The Kansas Academy of Family Physicians provided significant assistance in the preparation of these Guidelines.

Address reprint requests to Kansas Regional Perinatal Care Program, Kansas Dept. of Health & Environment, Topeka KS 66620.

2. An active and accessible program for the identification of actual or potential high risk pregnancies at the earliest possible date in the pregnancy, appropriate consultation with specialty physicians, and related antepartum management.

3. Physically separated facilities for obstetrics which does not preclude the use of the obstetrical unit for certain clean surgical cases, or the use of the postpartum unit for clean gynecological cases. Control of this use must be in accord with the requirements of the local infection committee.

4. Competence to manage uncomplicated labor and delivery of normal term newborn infants. Personnel in these Centers should maintain an awareness of new developments in technology and services offered at other Centers in the system and avail themselves of these services as indicated. Capabilities for continuous maternal and fetal electronic monitoring with external tocotransducer and fetal Doppler or scalp electrode are also recommended.

5. An active in-service (continuing education) program including recognition of complications in the mother, fetus and newborn, and related management strategies such as maternal and neonatal resuscitation. Each delivery room should have equipment and facilities as required by KANSAS HOSPITAL REGULATIONS, 6th Edition, 1974 (or most recent adopted regulations), Kansas Dept. of Health & Environment, Topeka KS 66620.

6. A defined routine of transitional stabilization, examination, immediate care, and surveillance of all newborns.

7. A nursery able to provide supportive care for infants transferred back from Level II or III Centers *after* their acute problems have been resolved. Continued surveillance and family centered care can well be provided in the original or local hospital.

8. Management of unexpected complications. Services and facilities for emergencies should include:

—Cesarean section capability within a short start-up time consistent with good medical care. Each hospital should establish a protocol to minimize start-up time for cesarean section.
—Blood available on call on a 24-hour basis. Type O, Rh negative blood should be readily available at all times.
—Anesthesia service on call on a 24-hour basis, with a response time consistent with protocol established for cesarean sections.
—Radiology service on call on a 24-hour basis, including capability to perform basic radiologic studies for women and infants.
—Clinical laboratory service on call on a 24-hour basis, with capability to perform the following semi-micro studies necessary to basic newborn care: CBC, type and cross match, glucose, bilirubin, serum calcium, Coombs' test, CSF protein, and cell count within one hour of the time specimen is received. Capability to perform bacterial cultures should also be available.
—Capability to initiate intravenous therapy in a newborn.
—A special care area should be available in which severely ill infants may receive further treatment prior to transfer to a Level II or III Center. Capability in this nursery should include short-term critical care.

9. Uncomplicated postpartum care, including postpartum recovery and observation area for mothers and newborns.

10. Management of unexpected postpartum complications. Capabilities must include isolation facilities for mother and newborn (see no. 3); blood available on a 24-hour basis; bacteriologic diagnosis (blood culture, etc.); clinical laboratory service for newborns listed above; and x-ray diagnosis for mother and newborn.

11. Perinatal data collection for evaluation of function and performance, locally and regionally, including cesarean section performance.

12. A system to facilitate coordination with the County Health Department for prenatal and postpartum referral for mothers and infants at risk.

13. Family-centered maternal/newborn care and policies that will promote and maintain family wellness and relationships and provide preventive elements for positive parenting.

14. Provision for patient/family perinatal education that will emphasize the normal processes of pregnancy, labor, delivery, and the postpartum period; preparation for childbirth; maternal nutrition; infant nutrition and feeding; newborn care; parenting skills; and general health maintenance.

Criteria

The criteria for consultation, evaluation, and possible transfer of high risk pregnant women and newborns are:

1. Early recognition of potential high risk patients is necessary for appropriate consultation and possible transport to a Level II or III Center. Consultation may consist of telephone discussions or antepartum/intrapartum visits to or by the consultant.

2. Each hospital should have a working relationship with a Level II and Level III Center for consultation and transfer when necessary.

3. When perinatal problems which may require transfer of the neonate are anticipated, maternal-fetal transfer should be considered rather than delivery in a Level I Center.

4. Transfer of a patient should be preceded by physician-to-physician communication in all cases if at all possible.

Personnel

The personnel requirements in Level I Centers are as follows:

1. Consultation. To facilitate and speed consultation, it is necessary to have specialists from Level II and Level III Centers available as consultants to the Level I Center.

2. Administration and supervision of nursing services. Every Level I Center should have a registered nurse whose primary responsibility is the organization and supervision of nursing services in the labor/delivery area, the newborn nursery, and the postpartum units. The nurse should possess knowledge and clinical competence in nursing care of mother, fetus, and infant during labor and delivery, and in the postpartum and neonatal periods. One or more physicians and the head nurse should develop operating policies and maintain standards for care of neonatal services in consultation with the network's Level II and III hospital staff.

3. Nutrition services. A registered dietitian/nutritionist should be available for consultation and for providing in-service and outreach training. Nutrition consultants and other health care team members should have experience in nutritional management and counseling regarding the nutrition needs of pregnant women, normal newborns and lactating mothers, and should be aware of community nutrition resources.

4. Labor and delivery. All deliveries should be attended by a physician. The physician should examine the mother at appropriate intervals during labor to assess her condition and the status of the fetus. The physician should be immediately available during the later stages of labor or when there is evidence of fetal distress or maternal complications. The responsible physician must be present in the labor area during the initiation of oxytocin infusion to manage unexpected maternal and fetal responses. Following this initial period, the physician must be immediately available to respond in case of emergency during administration. Intravenous infusion of oxytocin for induction or labor augmentation should be performed with a double infusion arrangement and oxytocin infusion pump.

If a high risk mother is to be (unavoidably) delivered at this level, every attempt should be made to have a neonatal oriented physician available at delivery. In case of emergency cesarean section or unexpected fetal distress, a second physician should be available to assist the primary physician with the care of the mother and subsequent resuscitation and care of the newborn. If this is not possible, the physician may be assisted by a surgical technician. The registered nurse serving in the delivery room must be competent in resuscitation of the newborn.

Adequate anesthetic coverage should be provided and supervised. Safety of obstetric anesthesia depends principally on the skill of the anesthetist. The same level of competence of anesthesia personnel should be required for obstetrical procedures as for surgical procedures.

In addition to the physician involved in the delivery and the anesthetist or anesthesiologist when needed, the delivery room should be staffed with at least one registered nurse capable of caring for the infant following delivery and assisting in the care of the mother. In addition to a registered nurse, a nurse's aide or obstetricial technician should be available to assist with those tasks that might divert the nurse from immediate care of the mother and infant.

There shall be two or more qualified* registered nurses available on call to be in the hospital if a woman is admitted in labor. Such nurses should remain in attendance during delivery and the immediate recovery period.

A registered nurse may be made responsible for initial evaluation and admission of a woman in labor. S/he may be delegated responsibility for continuing assessment and evaluation of the course of labor, the status of the fetus, and for determining the presence or absence of complications. A licensed practical nurse or nurse's aide, supervised by the registered nurse, may provide personal support of the mother and attend to her personal comfort and needs. Responsibility for following the course of labor or the status of the fetus may not be delegated by the physician to anyone except the registered nurse.

5. Early postpartum period: *Infant*. An initial evaluation of every newborn should be carried out by a physician responsible for the care of the infant

* For the purposes of these Guidelines, a qualified registered nurse must be an R.N.; meet continuing education requirements as stipulated by the Kansas Board of Nursing; attend courses relating to maternal and child health nursing; and be regularly assigned to the maternity unit to provide consistency in practice and care.

or a registered nurse with training and experience in the recognition of abnormalities in the newborn. Supervision of the infant through the period of autonomic neurological adjustment and recovery from the delivery process until the infant is stabilized and has successfully taken feedings is the responsibility of the registered nurse. This responsibility may be delegated to a licensed practical nurse or other qualified nursing technician under the supervision of the registered nurse who is on the premises.

This recovery period for the newborn, the nursing examination and serial observation of vital signs should be performed according to a clearly delineated protocol established by the medical and nursing directors of the nursery. A process of "graduation" from this recovery period to well newborn care should be similarly defined.

The registered nurse is responsible for notifying physicians of any abnormalities or problems in the newborn when they are detected. Infants with abnormalities requiring transport to another hospital should be directly supervised by the registered nurse during the period of medical management while they are awaiting transportation.

Maternal. The mother's care during the immediate recovery period following delivery should be supervised by a registered nurse, preferably the nurse involved with the care of the mother during labor and delivery. A protocol for observation is required. This responsibility may be delegated to a licensed practical nurse or other qualified nursing technician under the supervision of a registered nurse who is on the premises.

6. Later postpartum period: *Infant.* The newborn infant should be viewed as stabilizing during the first two hours of neonatal life. A highly skilled registered nurse should supervise neonatal care and observation during this time. Under no condition should an infant be left unattended during this period of stabilization. A qualified, supervised licensed practical nurse or a qualified nursing technician under supervision of a qualified registered nurse may provide care to the normal newborn. Under no condition should an infant be left unobserved during nursery care. This care should be consistent with the GUIDELINES FOR PERINATAL CARE, American Academy of Pediatrics and American College of Obstetricians and Gynecologists, 1983.

Rooming-in arrangement with adequate nursing assistance to the mother should be encouraged.

Maternal. The nursing care of postpartum patients should be directed and supervised by a qualified registered nurse. Licensed practical nurses and qualified nursing technicians may provide care for patients without complications and, under supervision, for patients with complications.

The support of mothers in their relationship to their infants is a very important role which must be carried out by a competent nurse. Postpartum care should also include education of the mother to care for her infant.

Discharge Planning

Discharge plans should be developed in cooperation with the patient/family and include referral as needed to community nursing, food/nutrition assistance and education, parenting education, and income maintenance resources to assure a positive maternal and newborn health status.

Resource: Related Documents

Guidelines for Perinatal Care, American Academy of Pediatrics, P.O. Box 1034, Evanston IL 60204, or American College of Obstetricians and Gynecologists, 600 Maryland Avenue SW, Suite 300 East, Washington, DC 20024; 1983.

Toward Improving the Outcome of Pregnancy, National Foundation-March of Dimes, 1275 Mamaroneck Avenue, White Plains NY 10605; 1976.

Ambulatory Maternal Health Care and Family Planning Services, American Public Health Association, 1015 18th Street NW, Washington DC 20036; 1978.

Development of Family-Centered Maternity/Newborn Care in Hospitals, Interprofessional Task Force Secretariat, ACOG, One East Wacker Drive, Suite 2700, Chicago IL 60601; 1978.

Nutrition Services in Perinatal Care, National Academy Press, Washington DC; 1981.

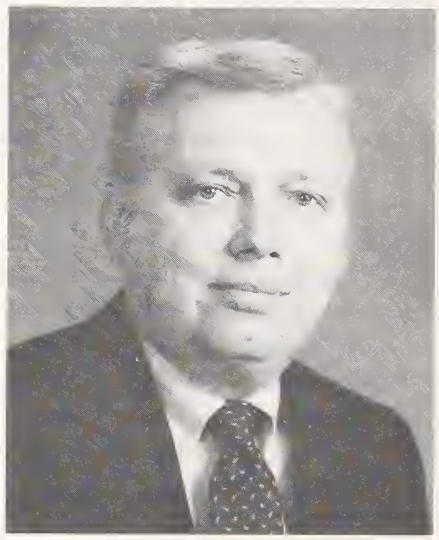
Standards for Obstetric, Gynecologic, and Neonatal Nursing, The Nurses Association of the American College of Obstetricians and Gynecologists, 600 Maryland Avenue SW, Washington DC 20024; 1981.

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Tucker, Susan Martin, RN, BSN: *Fetal Monitoring and Fetal Assessment in High-Risk Pregnancy,* The C. V. Mosby Company, St. Louis; 1978.

Young, Diony: *Changing Childbirth Family Birth in the Hospital,* Childbirth Graphics Ltd., Rochester; 1982.

The President's Message



The year 1983 has been a memorable one. Evaluation in three to five years will determine if the events of this year were good or bad. Some of the events were:

- The DRG system was established for Medicare. A Blue Cross-Blue Shield DRG system will be initiated January 1, 1984;
- The Blue Cross-Blue Shield contract became effective September 15th;
- HMOs, PPOs, IPAs proliferated;
- Networks of hospitals increased throughout the state;
- The Health Care Stabilization Fund has been evaluated and is being revised;
- The State Supreme Court ruled that records of hospital committees are no longer privileged.

All of the above issues created a great deal of discussion, anxiety, and frustration for all of us. The continued attention to these matters will consume much of our time in succeeding years. Only an active and well informed medical community will be able to deal with these problems in the future.

We wish to express our sincere appreciation to:

- Steve and his marvelous staff who have worked so hard and long for all of us;
- The executive commitee, councilors, committee chairmen, and committee members for their long hours of study, deliberation, and faithfulness to their tasks;
- The KMS Auxiliary for their interest in aging and many other projects;
- And to you, the members of this society, who have given us the spark to continue to be involved in the many issues that detract from the practice of medicine.

We pledge to you a continuation of our total effort to keep you informed of the issues, to be active in the legislature on your behalf, and to work on fiscal and economic problems.

We extend our greetings to you and your family for a Happy Holiday Season. Best wishes, too, for a Prosperous and Happy New Year.

Jimmie A. Pearson M.D.

President



Editorial COMMENT

A beggar to the graveyard hied
And there "Friend, corpse, arise," he cried.
"One moment bear my heavy weight
Of poverty for I of late
Grow weary and desire instead
Your comfort — you are good and dead."

The corpse was silent — he was sure
'Twas better to be dead than poor.

It is doubtful that poverty as experienced by the Editorial Board is on a par with that known to the ancient Hindu who composed those lines (as translated by Arthur W. Ryder in *The Panchatantra*). Moreover, as recently as the Board's latest meeting, there were no overt expressions of death wishes although the Editor was heard to make certain guttural sounds of distress when his colleagues cheerily instructed him to implement certain actions they had so glibly devised.

It comes down to a deficiency state to which the *Journal* has had some susceptibility over the years, characterized by nummular paresis and redness of figure. It seems not too long ago we were assuring the membership of our certain although fragile solvency. Furthermore, we naively reported that we would gladly relinquish to the Business Manager the duty of informing the membership if our fortunes changed (and Steven, where *are* you?)

Our story is not unusual in the current economic climate but we can't call on the government for help *a la* Chrysler or blame labor's excesses as do the airlines. We might make a case for having to contend with foreign imports but we'll certainly never admit to poor stewardship so we're faced with the same tired excuse — inflation has caught up with us. Whatever the mechanism, the outgo has been exceeding the income and there is a limit to that sort of thing without an act of Congress.

So the Board has been meeting in solemn session with unusual frequency in an effort to devise appropriate measures to stay afloat. Possible solutions (at least those that can be reported in a family type magazine such as this) have ranged from the hopeless to the ridiculous but come back to the basic problem — cutting publication costs by reducing the content without changing the character of the Board's favorite journal. As you'll learn to your regret, we have conned the Council into a subscription increase but not without a firm parental admonition to make with the austerity. Still, we recognize there is something different in the present situation. It has to do with communication techniques going far beyond the columns of the *Journal* into the enveloping economy of the day. The "fixes" of the past no longer seem to fix, at least for very long. The year 1984 is here but bringing a world even George Orwell didn't foresee in its full implications. But more of this another time.

Well, it is reliably reported by horticulturists that rigorous pruning often brings new life to the prunee. We hope that will be the case, but in the meantime certain changes will be evident. Some of the features we have been pleased to publish in the past will have to be eliminated or reduced in frequency. The most difficult area will be reducing articles to the shortest form compatible with their message and the author's blood pressure. One of the characteristics of writing is that, as the project progresses, the writer develops an increasingly proprietary attitude toward it and any intrusion, particularly any threat of abbreviation, is an affront at least on a par with violating Mom while she is baking that pie and waving the flag. In short, the Board will be approaching with more vigorous zeal its twofold obligation: determining the optimum form of the reduced structure — and accomplishing it. Consequently, prospective contributors are advised that brevity will be the soul of appeal in their offerings.

One of the more noticeable changes will be the absence of references. Our reasoning is that (1) they take up a lot of space, and (2) readers aren't going to have the sources at hand so, if they won't take the authors' word for it, they can get them from the authors — who will thereby have a more positive indication of interest than those occasional requests for reprints.

Since there has been no greater offender in space run-overs than the Editor, he now, in the greater-love-hath-no-man tradition, acknowledges a higher law (how are we doing, Steve?) and dedicates himself to curbing his characteristic verbosity lest he find himself cut off in midsente —*G.*

Dear Colleague:

In a little less than a year from now, we will have a newly elected group of public officials.

It is crucial to the democratic process that each of us actively participate in their election — and it is our duty.

More than this, many of the key issues faced by these officials will deal with our practice and our freedoms to a degree undreamed of only a few short years ago. We have every right to speak up constructively on behalf of the common interests of both our patients and ourselves.

The most effective way to "speak up" is to join our medical society, work in its committees, and contribute to KaMPAC for 1984.

Your 1984 KaMPAC contribution — $100 for sustaining membership or $60 for regular membership — is now payable.

Please help! The price of freedom is eternal vigilance — and a contribution to:

KaMPAC
1300 Topeka
Topeka KS 66612

Roger D. Warren, M.D., *Chairman*
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CONTRAINDICATIONS: Hypersensitivity to any of the components, concurrent MAO inhibitor therapy, severe hypertension, bronchial asthma, coronary artery disease, stenosing peptic ulcer, pyloroduodenal or bladder neck obstruction. Do not use in children under 12 years.

Do not use this drug in patients with narrow-angle glaucoma, obstructive or paralytic ileus, intestinal atony of the elderly or debilitated patient, unstable cardiovascular status in acute hemorrage, severe ulcerative colitis, toxic megacolon complicating ulcerative colitis, myasthenia gravis. Do not use in nursing mothers.

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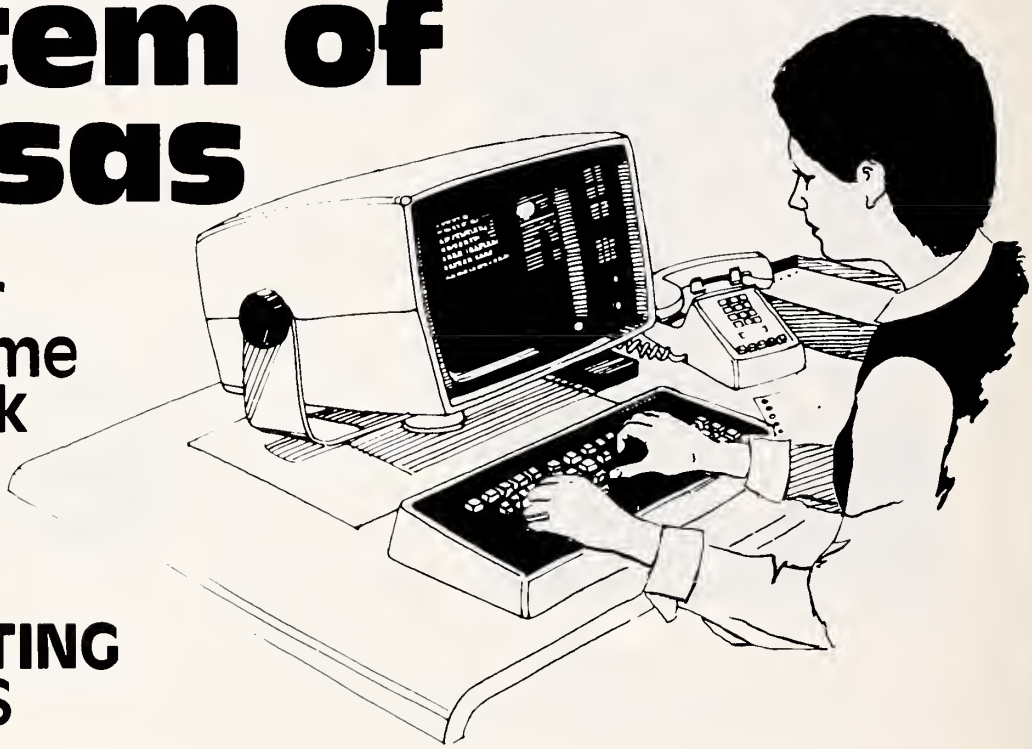
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Cardiac Pacemakers

(Continued from page 598)

ing an "atrial kick" to the cardiac cycle.[5] Atrial contractions increase the left ventricular end-diastolic pressure, and by the Frank-Starling mechanism, the longer muscle fiber length improves cardiac output. Dual chamber pacing enables younger patients or those with an active life style to achieve a better exercise tolerance by increasing the ventricular rate as the sinus rate increases with exercise. Some patients who have decreased myocardial compliance or myocardial dysfunction secondary to various cardiomyopathies may benefit from the increase in cardiac output with dual chamber pacing.

Dual chamber pacemaker units have different characteristics and advantages. In general, they require the use of two endocardial leads. One lead is the usual ventricular endocardial lead. The other lead is placed in the atrium or the coronary sinus. The most commonly used lead is the atrial J lead, whose curve allows it to be positioned in the atrial appendage. This can be used for sensing or pacing of the atrium. One type is the DVI (A-V sequential) pacemaker which consists of a unit that senses in the ventricle, and then paces the atrium and the ventricle synchronously based on the ventricular rate. This may be useful when there is prolonged retrograde conduction, but it does not allow for an increased heart rate with exercise. The VDD unit (atrial synchronous) is useful in patients with normal sinus node function and AV block. Since this unit paces the ventricle at the patient's own sinus rate, it allows the heart rate to increase with exercise. The newest unit is the DDD (A-V universal) pacemaker, which combines properties of the DVI and the VDD units. The use of dual chamber pacing is not a panacea, however.[6, 7] Patients who have some types of sinus node dysfunction, prolonged retrograde conduction, and the possibility of atrial flutter/fibrillation may not be safe candidates for dual chamber pacing.

Summary

Permanent transvenous cardiac pacemakers are safe, and many improvements have been developed. With newer pacemaker units and implantation techniques, morbidity and complications should be low. Improved cardiac performance may be achieved in some patients using dual chamber pacemakers. Finally, pacemaker followup, chronic threshold measurements, and reprogramming should increase pacemaker longevity and make permanent cardiac pacing even safer.

References are available from Dr. Bosiljevac, 2522 W. 15th Street, Emporia KS 66801.

Biliary Cysts and Carcinoma

(Continued from page 601)

Case Report

A 27-year-old white female presented with a history of obstructive jaundice at age 17 years, and laparotomy revealed a Type I biliary cyst which was treated with a Roux-en-Y choledochocystojejunostomy. Postoperatively, an anastomotic stricture required revision less than one year later. She suffered right upper quadrant pain during her second pregnancy at age 23 years. This pain resolved spontaneously postpartum, and she remained asymptomatic until the pain recurred three months prior to admission. Three weeks prior to operation she developed nausea, vomiting, fever, and chills consistent with cholangitis. Percutaneous transhepatic cholangiogram demonstrated a dilated biliary system with a stricture of the common duct above the choledochocystojejunostomy. Physical examination revealed an obese white female with mild right upper quadrant tenderness without peritoneal signs.

At laparotomy a hard mass involved the anterior portion of the cyst and encased the portal vein and hepatic artery. The tumor was debulked, and a transhepatic U-tube placed through a new choledochocystojejunostomy. Her postoperative course was complicated by wound dehiscence, which was satisfactorily repaired. She was discharged six weeks after the first procedure with normal liver function studies including a total bilirubin of 0.9 mg/dl.

The pathologic report revealed a moderately well-differentiated adenosquamous carcinoma, involving the soft tissues surrounding the cyst and at the resection margins.

Unfortunately, she suffered a progressive downhill course with 22.7 kg weight loss, nausea and vomiting, and died four months after discharge.

References are available from Dr. Wade, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.

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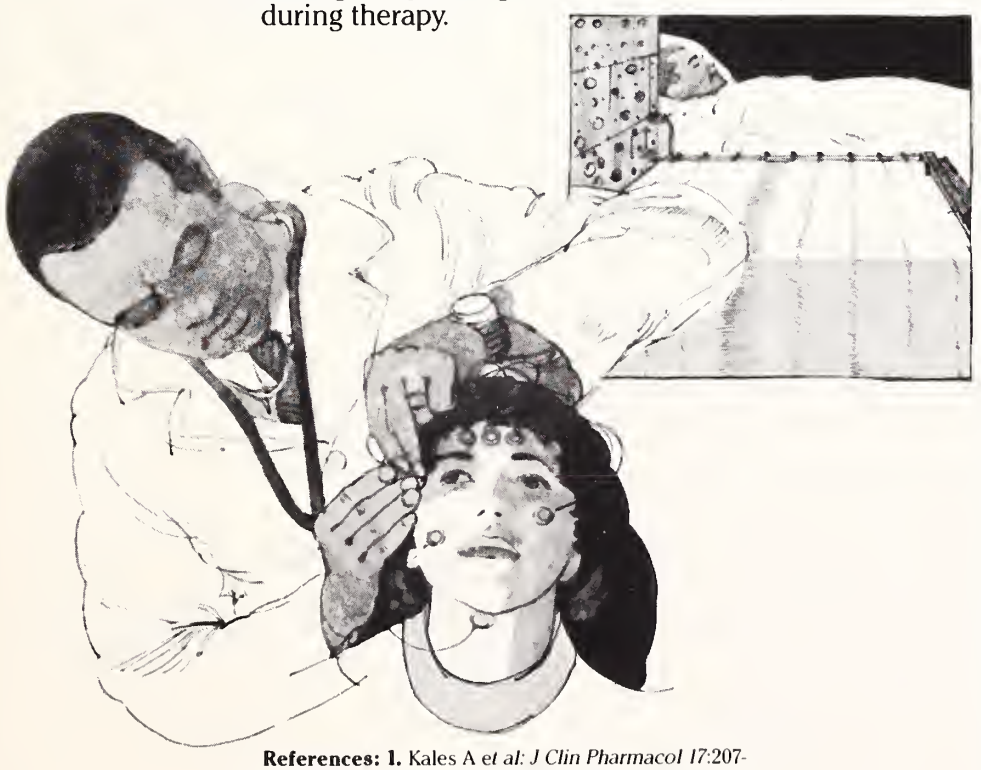
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